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Technical Report
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H.C. Hughes, P.A. Reuter-Lorenz, R. Fendrich,
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Program in Cognitive Neuroscience

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Multimodal Interactions in Sensory-Motor Processing

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ABSTRACT

The saccadic control system represents a good model system to study the selection of stimulus events according to their spatial location. The present work focuses on two factors known to influence saccade latency: the presence of a fixation stimulus and the nature of the saccade target. We report evidence which suggests that fixation point offsets facilitate pre-motor stages of saccade generation (Reuter-Lorenz et al., in press; Appendix I). This idea, in conjunction with electrophysiological data, suggested that fixation offset might also facilitate saccades to acoustic targets. Experiment 1 confirmed this suggestion (Fendrich, et al. [in preparation]). The facilitatory effects of redundant stimulation via the visual and auditory modalities is examined in Experiment 2 (Nozawa et al., 1990).The data suggest significant neural summation, which we attribute to bimodal convergence onto individual cells thought to mediate saccadic command functions. Finally, analytic methods for establishing these conclusions (Nozawa, Appendix III) and progress on computer-aided reconstruction of lesion sites in humans (Tramo et al., Appendix II) are described. The detailed analysis of lesion site represents a prerequisite to future efforts to identify the neural structures which mediate these influences over oculomotor efficiency.

Introduction

In natural situations, organisms must be able to process sensory information originating from many different spatial locations. Since any information processing system has capacity limitations, there is a need for a "selection schedule" whereby different spatial locations are sampled serially. While there may be instances in which spatially distributed sensory information can be processed in parallel, there are a great many situations in which serial processing is required. One clear example of serial processing in vision is the sequence of fixations observers make when inspecting a complex scene. Since the greatest degree of spatial resolution is confined to the area centralis or fovea, which represents only 0.01% of the visual field (Carpenter, 1977), there is the need for an elaborate motor system capable of quickly moving the eyes from one area of interest to another (i.e., the saccadic control system) and a system for foveating objects in motion (i.e., the slow pursuit system). It is hardly coincidental that the development of the oculomotor system attains its greatest elaboration in those mammalian species who also have well-developed foveas. Thus, eye position indicates the portion of the visual field currently being processed with the greatest precision, and the fixation point is often taken to indicate the current locus of visual attention.

It would appear almost axiomatic that overt shifts of eye position should be under multimodal control, since objects located in the visual periphery are likely to produce stimulus energy in more than one modality. Thus, a visually camouflaged object might be detected first in the auditory modality. This auditory input could elicit a shift in eye/head position which brings the image of the object into central vision, where the camouflage is more likely to be broken. However, very little information concerning the role of auditory inputs in controlling eye position is available (see however, Grusser, 1983; Mather and Lackner, 1980).

The discovery that the superior colliculus of the midbrain (SC) receives auditory inputs (Meredith & Stein, 1983; Peck, 1987; Wise & Irvine, 1983) suggests one possible mechanism whereby acoustic stimuli might generate saccades. There is strong evidence that the superior colliculus plays an important role in the generation of saccades: it receives visual input from both the retina and cortex, has efferent projections to pre-oculomotor brainstem structures, and can trigger saccades when focally stimulated (e.g., Sparks, 1986; Sprague & Meikle, 1965; Wurtz & Albano, 1980). Thus, the auditory input to the colliculus represents a subcortical link between the auditory and oculomotor systems.

The speed with which a stimulus can elicit a saccade is also an important parameter of oculomotor control: it is obviously desirable that the oculomotor system generate saccades as quickly as possible. In general, the latency to initiate a saccadic eye movement to a peripheral target is very similar to the latency to initiate a simple manual response to the same target (e.g.., Carpenter, 1977; Hughes and Kelsey, 1985). Under certain circumstances however, saccades can be generated with extremely short latencies (100-120 ms as compared with 200-250 ms for ordinary saccades or simple manual reaction times [RTs]). These short-latency saccades (referred to as *express saccades*) are enabled when the fixation point is extinguished 200 to 400 ms prior to the onset of the saccade target (e.g.., Fischer, 1987). While the mechanisms underlying the production of express saccades are not well understood (see discussion by Reuter-Lorenz, Hughes & Fendrich, 1990; included as Appendix I), it appears that the superior colliculus plays an essential role. Thus, ablation of the SC in monkeys permanently eliminates express saccades (Sandell, Schiller & Maunsell, 1984; Rohrer & Sparks, 1986). These same lesions serve only to increase the latency of normal saccades (Wurtz & Albano, 1980).

Since the SC is a structure that appears to be importantly involved in converting sensory signals to oculomotor commands, it functions as both a sensory and motor center. The results from an initial series of experiments supported by AFOSR Project # 89-0437 (Reuter-Lorenz, Hughes & Fendrich, Appendix I, in press) are interpreted in terms of a model of express saccades which emphasizes the pre-motor processes known to occur in the SC. In contrast to earlier views which attribute express saccades to enhanced processing of the visual target (Reulen, 1984a, 1984b) or the early disengagement of visual attention (e.g.., Fischer, 1987), Reuter-Lorenz et al. attribute the facilitatory effects of fixation point offsets to *enhanced pre-saccadic motor activity* in the deeper layers of the superior colliculus. This interpretation is based on the two principle findings reported in Reuter-Lorenz et.al.: 1). that the facilitatory effects of prior fixation point offsets (hereafter referred to as the "gap effect") on saccadic latency are additive with the effects of the luminance of the saccadic target and 2). the facilitatory effects of fixation point offsets is specific for saccades directed towards the target ("pro-saccades"). Tasks in which saccades are to be directed *away* from the target ("anti-saccades": See Hallett & Adams, 1980) and choice manual RTs are unaffected by extinguishing the fixation point prior to the delivery of the imperative stimulus. Thus, Reuter-Lorenz et.al. (in press) suggest that the response specificity is consistent with the view that the SC is essential for the occurrence of express saccades, since the SC contains circuits important for the production of pro-saccades but not anti-saccades or speeded manual responses. The additivity between the gap effect and the effect of target luminance supports the suggestion that the

gap effect operates at a "post-sensory" stage in the neural processes that translate sensory input into saccadic eye movements.

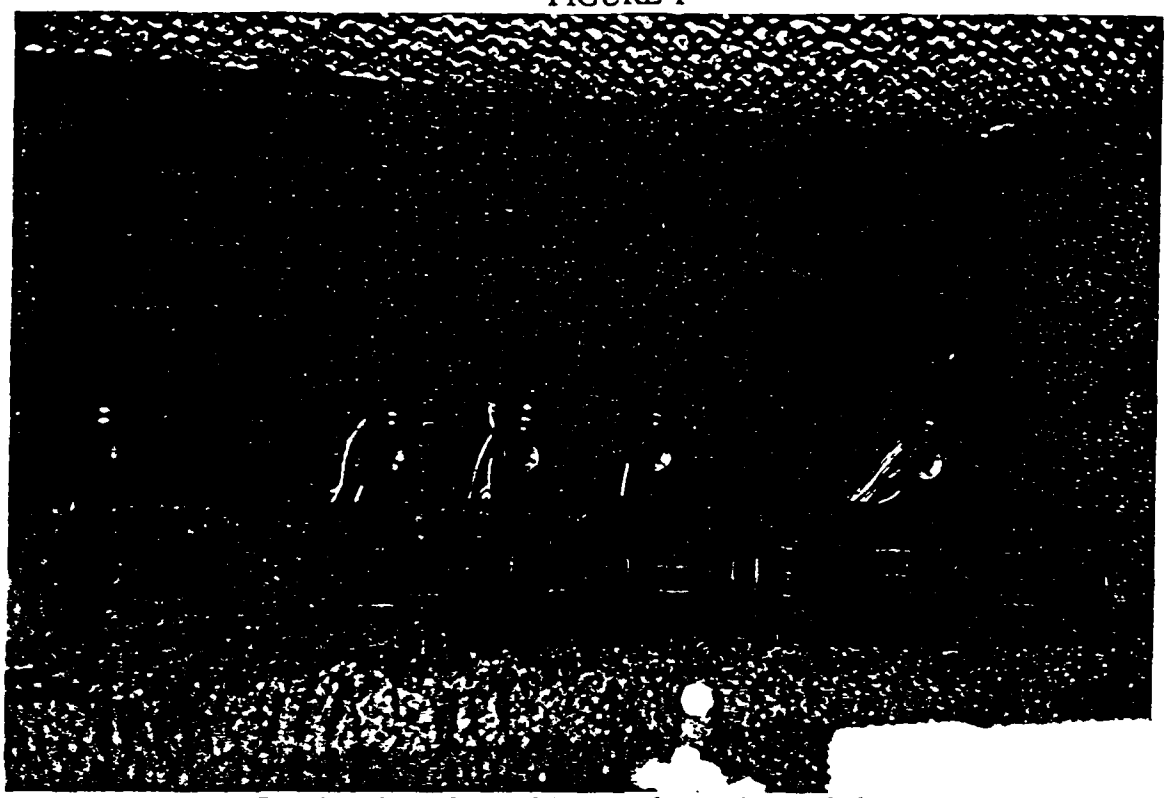
Thus, the mechanisms by which fixation point offsets facilitate saccadic latency and the functional character of multimodal convergence within oculomotor centers are clearly important issues in our continuing attempt to characterize the efficiency of oculomotor control. Here we report the results of additional work that derives directly from the considerations of oculomotor control outlined above. We first demonstrate that extinguishing the fixation point reduces the latency to initiate saccades for acoustic as well as visual targets. Thus, we establish that "express saccades" occur in the auditory as well as the visual modality. In a second series of experiments, we report on the facilitatory effects of multimodal targets on the latency to initiate saccades. As was indicated in our original proposal, true multimodal integration must be distinguished from latency facilitation produced by the simple summation of stochastically independent response times associated with each modality. We have made a great deal of progress in developing analytic methods to distinguish this *probability summation* from *neural summation*, and we outline the mathematical treatment of the data in Appendix III.

General Methods

Apparatus

The basic apparatus consists of an array of 5 stimulus panels aligned on an arc with a radius of 114 cm. Each stimulus panel contains a red light-emitting diode (LED), a green LED, and a small (4 cm) speaker. The position of each stimulus panel is adjustable. In the current series of experiments, the panels are positioned at eccentricities of 10^{o} and 20^{o} along the horizontal meridian of the left and right visual fields. The green LED of the middle panel serves as a fixation point and the red LEDs of the eccentric panels serve as the visual targets. Acoustic signals consist of brief (100 ms) bursts of white noise delivered through the speakers on the eccentric panels. Both the amplitude of the acoustic targets and the luminance of the visual targets are controlled by 12 bit D/A converters. In order to maintain equivalent degrees of observer readiness across conditions, acoustic warning signals (1000 Hz, 300 ms) presented through the center speaker precede the delivery of imperative targets. In order to prevent echoes which might impair sound localization performance, the entire apparatus is enclosed in a large (1.54 m. by 1.54 m. by .9 m.) enclosure which is lined with a sound-absorbing foam material (Sonex™). A photograph of the apparatus is shown in Fig. 1.

FIGURE 1



Interior view of sound-insulated experimental chamber.

Eye position is monitored using a scleral infra-red reflection device (Narco Biosystems™ Model 200 eye tracker). The output of the eye tracker is sampled via a 12 bit A/D converter at 250 Hz, and the digitized records are stored for subsequent off-line data analysis. In some experiments, saccade performance is compared to either directed manual responses or to simple manual reaction times (RTs). Directed manual responses are recorded using a inductive coil joystick, which is also sampled at 250 Hz. For this condition, subjects are simply instructed to move the joystick in the direction of the target's location as quickly as possible. Simple manual reaction times are recorded by depressing a microswitch, which is also sampled at 250 Hz. In this condition, subjects are instructed to depress the microswitch in response to any target as quickly as possible. Thus, all responses are timed with equivalent temporal resolution. The presentation of stimuli, timing of events and data collection are all computer-controlled.

The apparatus is located in an isolated, darkened room. Data collection is always preceded by 5 min of dark adaptation, during which time the eye tracker is adjusted and eye position is calibrated. Subjects view the display at a distance of 114 cm. Head movements

are minimized using a bite-plate. All subjects are emmetropic (or are appropriately corrected) and have normal hearing.

Response Detection. Both saccades and directed manual responses (joystick movements) are detected using a velocity criterion. While the detection of both saccades and joystick responses is automatic, an experimenter always monitors the records in order to insure that misses or false positives are not included within the data set. In general, the velocity criterion for saccades is set to ~ 50 deg. sec^{-1}, however, the criterion is sometimes adjusted in order to maximize the performance of velocity-based algorithm. This is especially true in the case of joystick responses, which tend to show greater variability than saccades.

Preliminary Testing. As might be expected, neurophysiological (Stein et al., 1989) and behavioral data (Miller, 1986) indicate that the magnitude of summation effects between two different modalities depend upon central simultaneity of the arrival times for the two inputs. Therefore we obtain extensive preliminary data comparing average response times to both visual and auditory targets in order identify stimulus energies that produce equivalent latencies in each subject. This is important because a large mismatch in the response times for each modality would minimize our ability to detect intersensory facilitation (the presumption is that the central arrival times from each modality should be nearly simultaneous since a large asynchrony between modalities will not produce the enhanced neural activity that underlies the effect).

The analytical methods described in Appendix III utilized the entire distribution of response latencies rather than simple comparisons of summary statistics, and therefore permit us to relax the requirement of central simultaneity. This not only maximizes the sensitivity of our measures of summation between modalities, but has the additional advantage of providing our observers with extensive practice on the various tasks prior to the formal collection of data.

EXPERIMENT 1 EFFECTS OF PRIOR OFFSET OF FIXATION POINT ON THE LATENCY OF SACCADIC EYE MOVEMENTS: A COMPARISON BETWEEN VISUAL AND ACOUSTIC TARGETS

Introduction

Extinguishing the fixation stimulus 200-300 ms prior to the onset of an eccentric visual target (the "gap paradigm") reduces the latency of a saccade directed towards that target (e.g., Saslow, 1967; Fischer & Breitmeyer, 1987). Fischer (1987) referred to these

short latency saccades as express saccades. The factors responsible for the production of express saccades are not yet understood. The latency reduction observed in the gap paradigm has been variously attributed to 1) enhanced processing of the visual target (e.g., Reulen, 1984a, 1984b), or 2) several possible efferent factors which ordinarily serve to delay the programming of saccades (Kalesnykas & Hallett, 1987; Saslow, 1967). A third possibility, that fixation point offsets facilitate the disengagement of attention and thus permit more rapid execution of the motor program has also been suggested (Fischer & Breitmeyer, 1987).

The findings of Reuter-Lorenz et al.(in press) were interpreted as arguing against sensory facilitation models. Rather, Reuter-Lorenz et al. suggest that fixation point offsets facilitate pre-motor processes. It is known that neurons in the deeper layers of the superior colliculus increase their rates of discharge 20-30 ms prior to the occurrence of saccadic eye movements. The activity of these "pre-saccadic burst" neurons is dependent on the particular vector of the ensuing saccade: each cell's burst is specific for a particular saccade vector. In addition, these cells increase their activity prior to "spontaneous" saccades made in darkness. These response characteristics suggests that the cells in the deeper layers of the SC are part of a motor command system for the initiation of saccades, an interpretation consistent with the fact that many of these cells project to an area of the pons known to control motoneurons of the extraocular muscles. Thus, Reuter-Lorenz et al. suggest that the occurrence of express saccades may rely to a large extent on the activity of the pre-saccade burst neurons in the deeper layers of the SC, a suggestion consistent with findings that lesions of the SC abolishes the occurrence of express saccades but not regular saccades (e.g., Sandell et. al., 1984).

Recently is has become clear the many of these cells receive convergent visual and auditory inputs (e.g. Jay & Sparks, 1990; Stein et al., 1989), suggesting a possible circuit whereby acoustic inputs can achieve control over the oculomotor system. Interestingly, there is good correspondence between the visual and auditory receptive fields of these cells, an obvious requirement of a system designed to direct the eyes to the source of either visual or auditory events. The interpretation of express saccades suggested by Reuter-Lorenz et al., in conjunction with the convergence of multimodal inputs on pre-saccadic burst neurons, naturally leads to the suggestion that fixation point offsets might facilitate saccadic latencies to auditory as well as visual targets. The present experiment is designed to evaluate this possibility.

Methods and Procedures

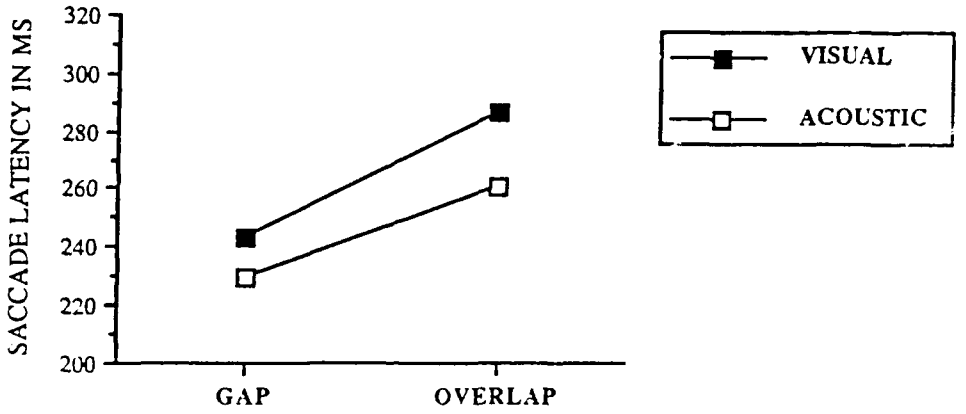
The design of the apparatus and the method of recording eye movements are described in the General Methods section of this report. The saccade target was 300 ms in duration and consisted of a white noise burst (90 dB) or the illumination of a red LED (0.7 cd/m^2). Pilot data indicated that these stimulus intensities produced approximately equivalent response times. The visual and acoustic targets were positioned at 10^o to the left or right of the fixation LED. Each block consisted of 72 trials. On half of the trials the fixation light (a centrally placed green LED) was extinguished 200 ms prior to the onset of the saccade target (gap trials) and on the other half, the fixation light remained on throughout the trial (overlap trials). To discourage anticipatory responding, 11% of the trials were catch trials in which no target event occurred. The remaining trials consisted of an equal number of acoustic and visual targets. Fixation condition and target modality varied randomly within each block. Six observers served as subjects. Each was an experienced subject in oculomotor experiments. Data were collected over the course of 7 experimental blocks. Each block consisted of 72 trials. Thus, each subject generated 112 observations for each of the four conditions (gap-visual target; overlap-visual target; gap-acoustic target; overlap-acoustic target).

Results

As previously reported by Jay and Sparks (1990), saccades to acoustic targets tended to occur in pairs in which the first and second saccade were of similar magnitude. Similar "double step" saccades are generated in the anti-saccade task (cf., Hallett & Adams, 1980; Reuter-Lorenz et al., in press), suggesting that double saccades might be characteristic of saccadic motor programs generated without visual guidance. The determination of saccade latency is of course based on the timing of the first saccade in such cases.

The average latency of the saccades obtained in each trial condition are illustrated in Fig. 2. It is evident that the latencies of both visual and auditory saccades was reduced by prior fixation point offsets. This effect was confirmed by an analysis of variance (ANOVA) on the individual subject means which indicated that only the main effect for fixation condition was significant, $F(1,5)= 12.98$; $p<.02$. The average magnitude of this "gap effect" was 42.5 ms for visually guided saccades and 31.5 ms for acoustically guided saccades. Although the magnitude of the effect appears slightly larger for the visual targets, the interaction term in the ANOVA is not significant, $F(1,5)= 4.49$; $p<.09$.

Figure 2



Discussion

The results provide a clear indication that fixation point offsets can facilitate the latency of saccadic eye movements triggered by auditory targets. The data therefore are commensurate with a model of express saccades which emphasizes premotor factors rather than sensory accounts of the gap effect. In particular, Reulen proposed that fixation point offsets exert their effects specifically within the early stages of the visual system, by facilitating the processing of visual events (Reulen, 1984a; 1984b). Together with the results of Reuter-Lorenz et al.(in press), the present finding of a gap effect for auditory targets is inconsistent with Reulen's account.

The attention based account of express saccades proposed by Fischer and Breitmeyer is also incompatible with the present observations. A number of studies have demonstrated that responses to auditory targets are unaffected by spatial precues, suggesting that prior attentional orienting is not necessary for detecting auditory signals (Buchtel & Butter, 1989; Posner, 1978). Thus, in the very least, the attentional disengagement hypothesized by Fischer and Breitmeyer (1987) appears to differ in important ways from that hypothesized on the basis of precuing experiments (see Appendix I for a discussion of this point).

The finding that fixation point offsets facilitate saccades to both auditory and visual targets is however consistent with the view that fixation release operates at a stage of oculomotor processing that is later than the stage which is common to both auditory and visual inputs. The functional nature of this convergence of auditory and visual information is addressed in Experiment 2.

EXPERIMENT 2. THE EFFECTS OF BIMODAL STIMULATION ON SACCADE LATENCY

Introduction

Even causal experience indicates that either visual or auditory events can elicit saccadic eye movements which serve to align the fovea with the source of the stimulus. Recent electrophysiological work has revealed a neural mechanism that appears designed to allow this multimodal control of saccades: individual neurons within the deeper layers of the superior colliculus receive convergent visual and auditory inputs (e.g., Jay & Sparks,1987; Peck, 1987; Meredith & Stein, 1987). The coordinates of the visual and auditory receptive fields are usually in spatiotopic register, and spatially aligned bimodal inputs often elicit unit discharges that are substantially more robust than the responses evoked using either modality alone (Meredith & Stein, 1987). Jay and Sparks (1987) report that many pre-saccadic burst (PSB) neurons receive convergent visual and auditory inputs, suggesting that the two modalities might show particular types of facilitatory interactions in controlling oculomotor responses. The present experiment represents an initial attempt to determine the degree to which spatially coincident auditory and visual targets can facilitate the latency to initiate saccades relative to the latencies associated with stimuli of either modality presented alone. The experiment thus addresses the issue of *intersensory facilitation* in the saccadic control system.

Central to a behavioral analysis of intersensory facilitation is the question of whether the facilitatory effects of bimodal stimulation are sufficiently robust to rule out the possibility that the bimodal responses times are simply determined by which ever modality is detected first (equivalent to the operation of a logical *OR* gate). As the detection times associated with each modality are themselves random variables, some reduction in responses times is expected in a system which applies such an OR operation to otherwise independent sensory channels, an effect known as *probability summation.* It is therefore essential to distinguish between true neural summation and the facilitatory effects that are accountable on the basis of simple probability summation. Our approach to this issue is being developed by Mr. G. Nozawa, a graduate student in Psychology at Dartmouth who is supported by AFOSR grant # 89-0437. The details of the mathematical treatment are presented in Appendix III of this report. In general, we compare the distributions of response times to bimodal stimuli to the distributions associated with unimodal stimuli using the laws of the joint probability of independent events. If the facilitatory effects of

bimodal stimulation exceed those predicted on the basis of the summation of independent events, simple probability summation can be rejected.

Methods and Procedures

Apparatus. The apparatus was the same as in the previous experiments, although the eccentricity of the targets was increased to 20 deg. of arc. Eye movements were recorded and analyzed as before. In addition to measuring saccade latencies to auditory, visual and bimodal targets, we included sessions in which the observers were required to generate manual responses under similar conditions. The purpose of these conditions was to compare the degree of intersensory facilitation for different response systems.

Two kinds of manual responses were examined. *Directed manual responses*, like saccades, required a response that was determined by the *location* of the target. Directed manual responses were recorded using an inductive-coil joystick. The subjects were simply required to push the joystick in the direction of the eccentric target as quickly as possible. Thus, these directed responses require location information (just as in saccades) but utilize a different response system. The joystick position was sampled using D/A convertors just as in the saccades (250 Hz sampling rate) and the direction and latency of the movements were analyzed in a similar manner. The second type of manual response was a simple reaction to the target onset. In this condition then, the location of the target is irrelevant to the performance of the task. Subjects simply depressed a microswitch in response to the target, and the latency of this response was the dependent measure.

Preliminary Procedures.

In order to identify stimulus intensities that produce equivalent latencies, observers were first run in a series of experimental sessions in which we measured their response latency as a function of stimulus intensity for both auditory (100 ms white noise bursts) or visual (red LEDs) targets. There were 64 trials in each session. Each trial began with a warning tone, followed by either a visual or auditory target (there were no bimodal stimulus trials in these preliminary sessions). Auditory and visual target trials were randomly mixed throughout the session, but were presented with equal frequency. Four different stimulus intensities were used for each modality, and the stimulus intensity and target location (left vs. right) also varied randomly across trials. Four of these preliminary sessions were run for each response system (saccades, directed manual responses, simple manual responses), so each data point of the obtained intensity-RT function is based on approximately 30 observations. The intensity-RT curves were used to select visual and

auditory stimulus levels which produced comparable response times for use in the formal portion of the experiment. This preliminary testing was done for each of the three responses to insure that the intensity matches were appropriate for each response condition.

Subjects.

To date three normal observers have participated in all phases of the experiment. Two others have participated in a subset of the conditions, but we report here only the data of subjects who have completed the experiment. Each was emmetropic or wore the appropriate optical correction. Each had normal hearing. The subjects were paid for their participation.

Formal Procedures.

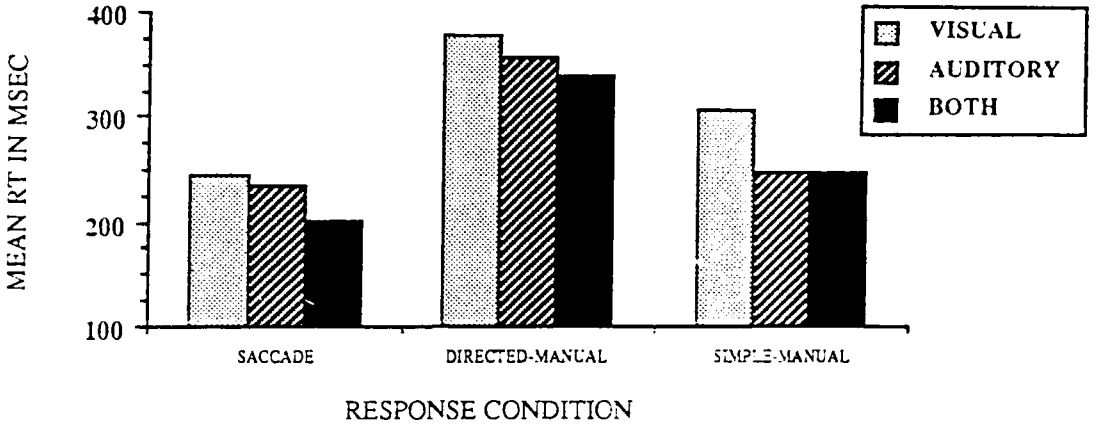
When intensities which produced equivalent latencies for the visual and auditory targets were identified, formal data collection began. Each observer participated in 15 experimental sessions of 60 trials each. Typically, a subject was tested for 2-3 sessions per day. Each session contained trials in which the imperative target was presented in either the auditory modality, the visual modality or both. Each target type occurred with equal frequency in a randomized order. In addition, the location of the targets (20 deg. on either side of fixation) was randomized. The data reported below are based on at east 100 observations for each stimulus condition (auditory, visual or bimodal) in each of the three observers.

Results

The averaged RTs for auditory, visual and bimodal targets are illustrated in Fig. 4. The left portion shows the saccade latencies, the middle shows the directed manual responses, and the bright portion shows the simple manual response times. It can be seen that for both types of directed response conditions, bimodal stimulation appreciably reduced response times. As pointed out in the Introduction to Experiment II, this reduction in response time could be accounted for on the basis of simple probability summation between independent visual and auditory signal processing. The important issue then, is to establish whether the magnitude of bimodal facilitation exceeds the level predicted on the basis of simple summation of independent sensory channels. If so, then the data cannot be solely attributed to probability summation. In this case, we should like to interpret the results as behavioral evidence of *neural summation* between the auditory and visual modalities. In the case of the saccade task, this summation is likely the result of convergence of visual and auditory information onto individual neurons importantly

involved in the generation of saccades; i.e., the bimodal pre-saccade burst neurons known to exist in the deeper layers of the superior colliculus. We now turn to an analysis that allows us to evaluate whether the degree of bimodal facilitation is greater than that attributable to probability summation alone.

FIGURE 4

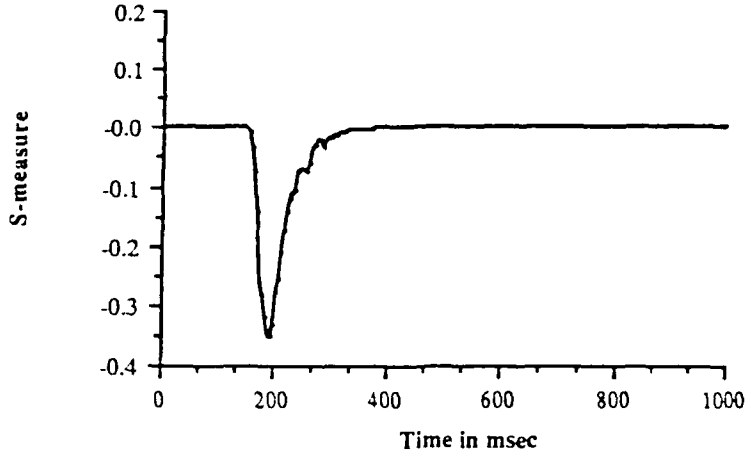


Evidence of Neural Summation based on Analysis of Survivor Functions (S measure)

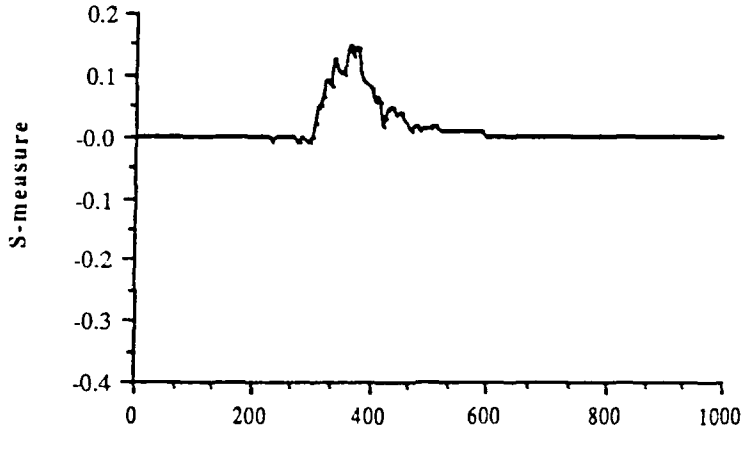
As described in Appendix III, we have two alternative measures of the expected magnitude of bimodal facilitation assuming statistical independence of the auditory and visual channels. First, we have the measure devised by G. Nozawa, termed the S measure. This measure is based on comparisons between the survivor functions obtained from the two unimodal conditions with that obtained in the bimodal condition (Eq 3 of Appendix III). If the bimodal survivor function is generated by a logical OR applied to the near simultaneous arrival of visual and auditory afferent activity (often called the "horse race model"), then the expected value of the S measure is 0. If the degree of bimodal facilitation is greater than that predicted by the horse race model, then the S measure assumes negative values. Our analysis therefore computes the S measure along the entire domain of the survivor functions (i.e., throughout the entire range of response latencies).

Obtained S measures for 3 subjects are shown in Fig. 5a -c. The top panel in each figure represents the data for saccades, directed manual responses are shown in the middle panel, and simple manual responses appear in the lower panel. It can be see that there is a clear ordering of the magnitude of the S measure between the three response conditions (saccades>directed manual responses>simple manual responses). The data provide clear indications that the magnitude of bimodal summation for saccades exceeds that predicted by

FIGURE 5a S1: Saccades



S1: Directed-Manual



S1: Simple Manual

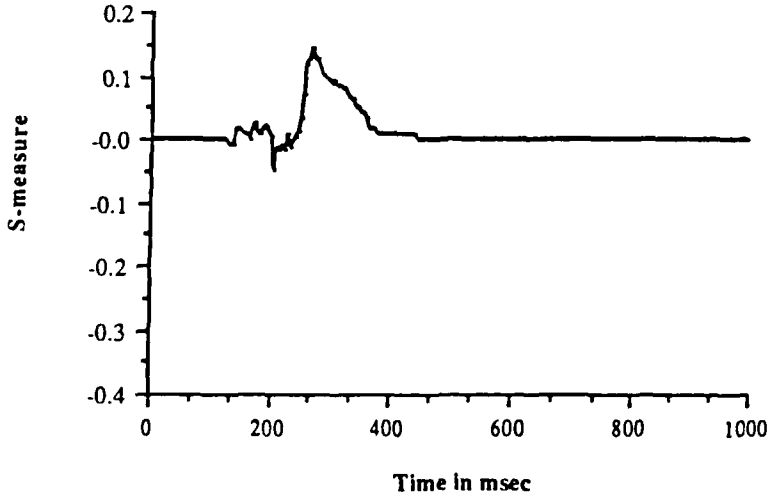
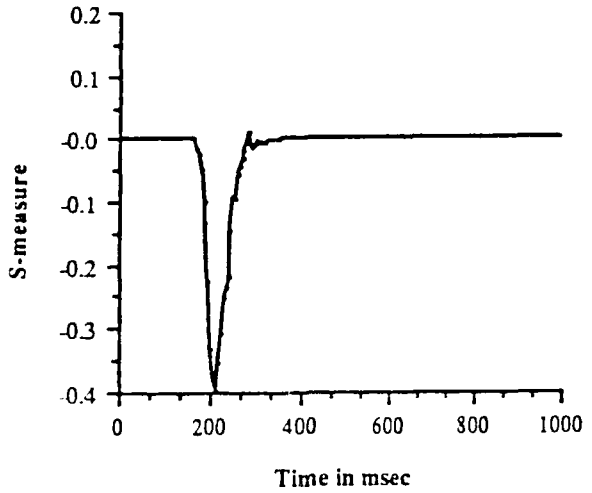
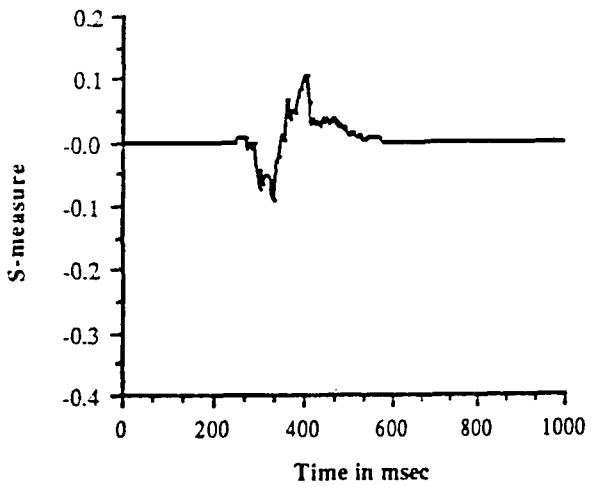


FIGURE 5b S2: Saccades



S2: Directed-Manual



S2: Simple Manual

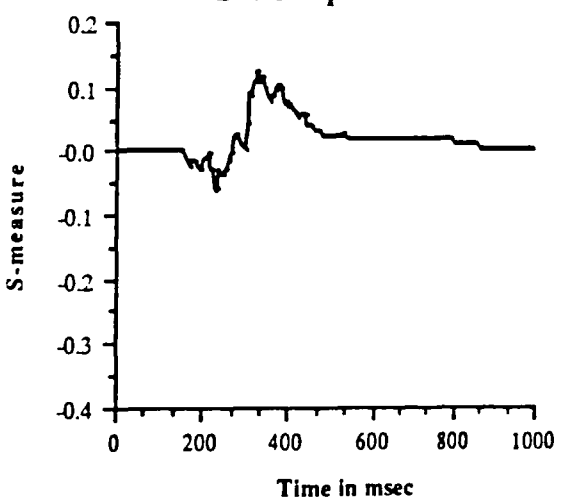
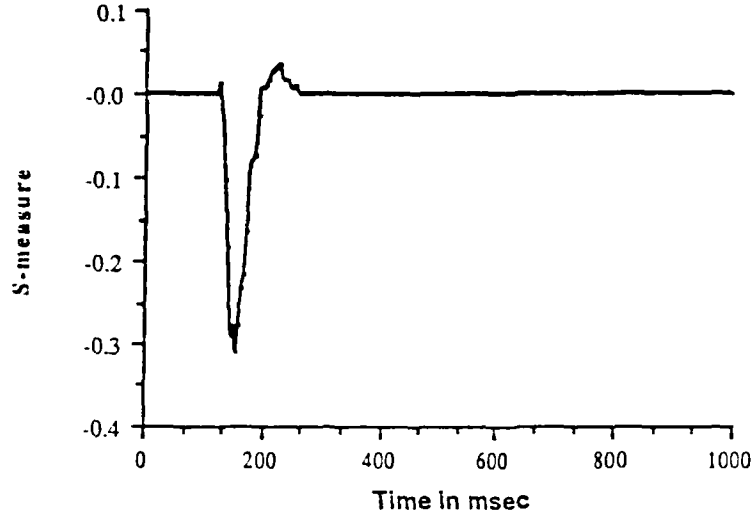
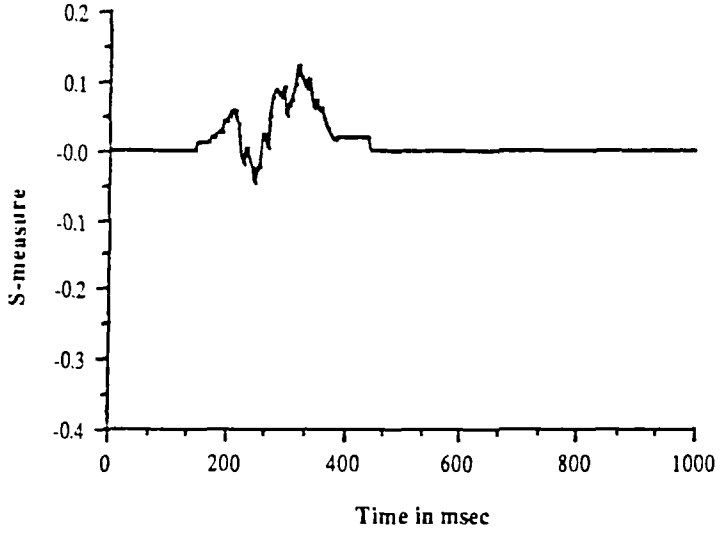


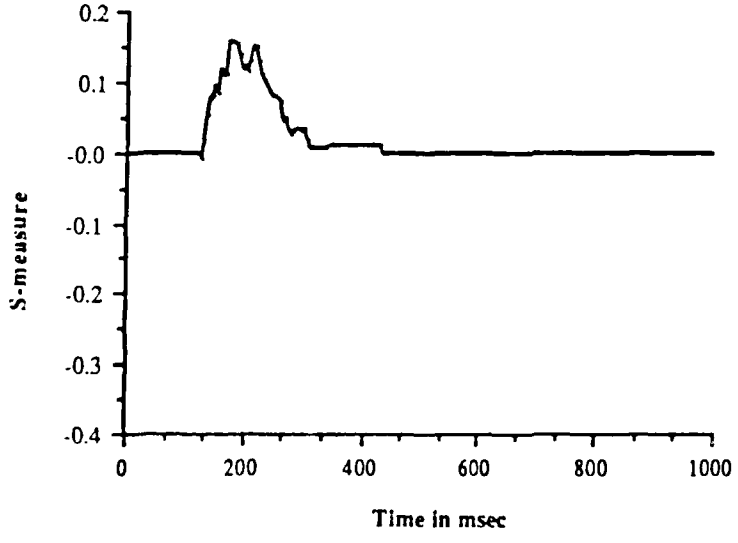
FIGURE 5c S3: Saccades



S3: Directed-Manual



S3: Simple Manual



probability summation, and therefore strongly suggest the existence of neural summation of the auditory and visual targets for this task. In contrast, directed manual and simple manual RTs provide little evidence of facilitation beyond that expected by probability summation alone (the S measure values remain near 0). This analysis shows that auditory and visual information probably do converge (in a facilitatory manner) onto neural mechanisms that play an important role in the execution of saccades. The results also provide little reason to invoke facilitatory neural convergence in order to account for the magnitude of summation particularly in the for simple-manual task. For two of the subjects (S2 and S3), however, there is a suggestion of some degree of neural summation for directed manual responses. It seems quite reasonable to suppose that the world of sensory interactions is not simply limited to two states (either probability summation or neural summation); various degrees of neural summation clearly seem possible. A method for distinguishing between levels of neural summation is being developed by a member of our group (G. Nozawa) and is outlined in Appendix III, so we should be able to apply tests of statistical significance to differences in the S measure in the near future.

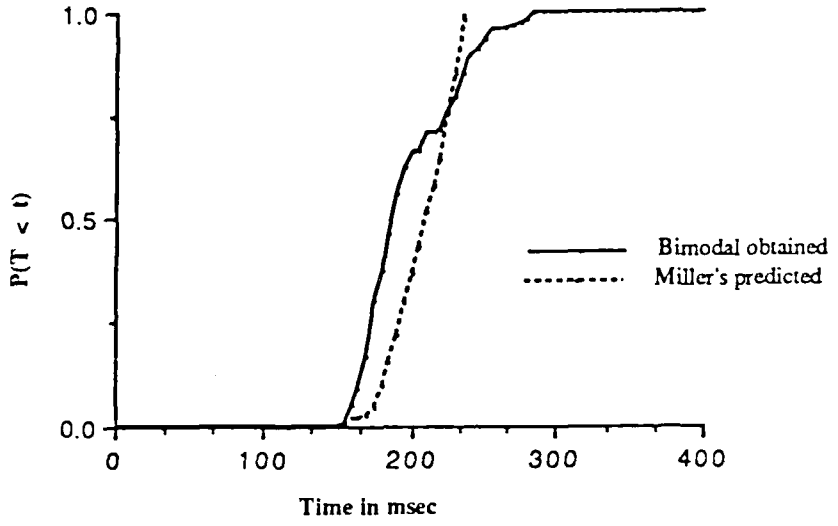
An application of Miller's inequality (Eq. 1 of Appendix III) is illustrated in Fig. 6a-c. In this case it is positive values provide the strongest evidence for neural summation between the auditory and visual inputs. It can be seen that the saccade data are consistently greater than zero. Thus, both measures indicate the existence of neural summation between visual and auditory channels in the generation of saccadic eye movements.

Discussion

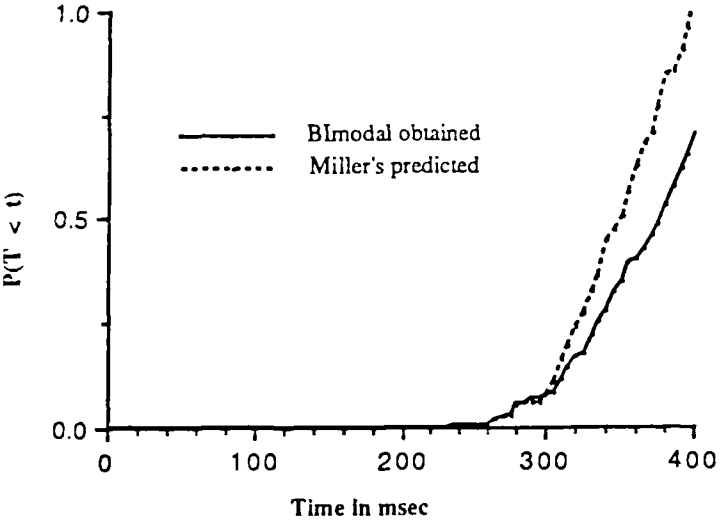
These performance data support the following conclusions: 1) the human oculomotor system combines localized auditory and visual information to a degree that is substantially greater than that expected according to simple probability summation; 2) the neural mechanisms that underlie directed manual responses and simple manual responses to bimodal stimulation may simply be triggered by whichever modality is detected first (logical OR applied to the inputs).

In the case of saccades, the most parsimonious interpretation is to suggest that the observed degree of intersensory facilitation is supported by the convergence visual and auditory afferents onto pre-saccadic burst neurons within the deeper layers of the superior colliculus (e.g. Peck, 1986; Jay & Sparks, 1987). This interpretation would receive strong additional support if it could be shown that intersensory facilitation of saccades depends on the spatial alignment of the visual and auditory inputs in a manner similar to that already described for the pre-saccadic burst neurons by Meredith and Stein (1987). Much of our future efforts supported by AFOSR grant # 89-0437 are designed to examine

FIGURE 6a S1: Saccades



S1: Directed Manual



S1: Simple Manual

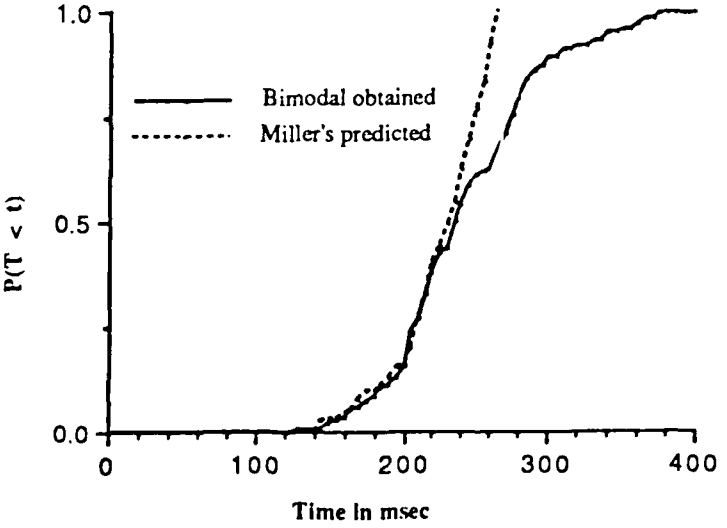
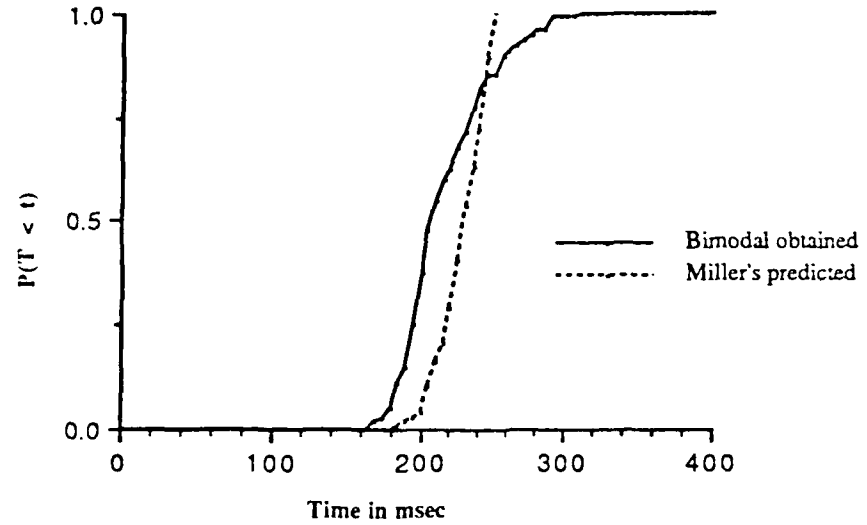
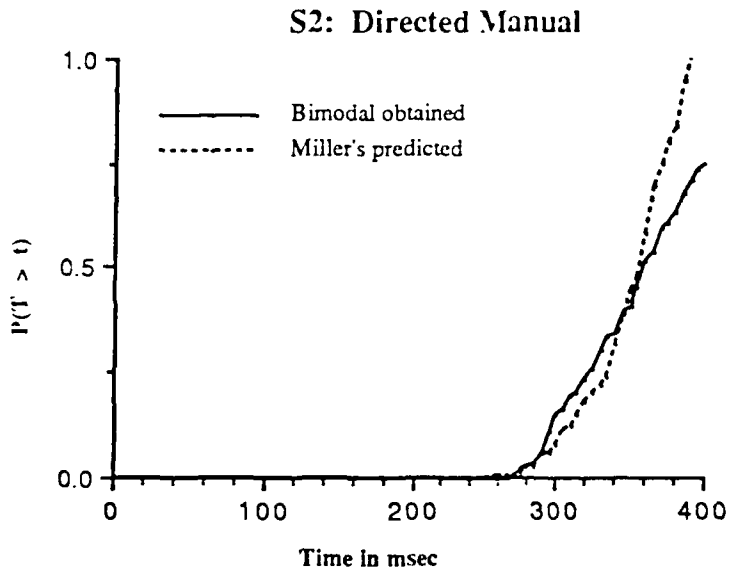


FIGURE 6b


S2: Saccades
1.0
0.5
0.0
P(T < t)
0
100
200
300
400
Time in msec
Bimodal obtained
Miller's predicted


S2: Directed Manual
1.0
0.5
0.0
P(T > t)
0
100
200
300
400
Time in msec
Bimodal obtained
Miller's predicted

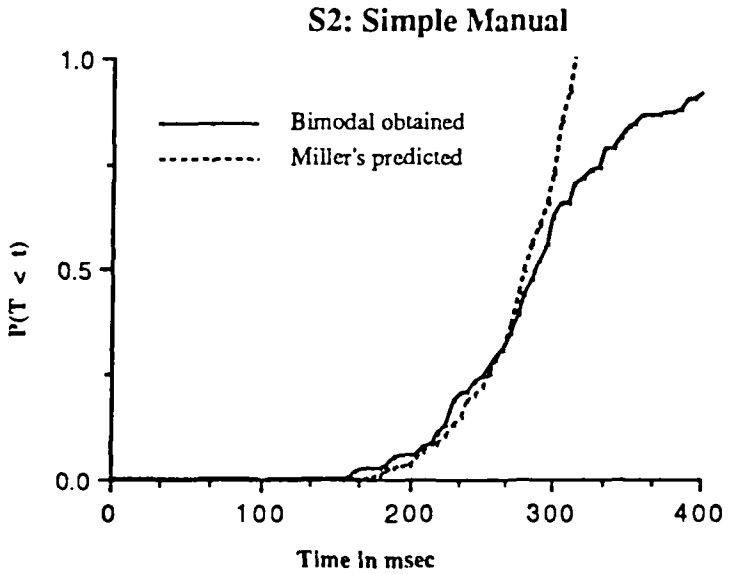
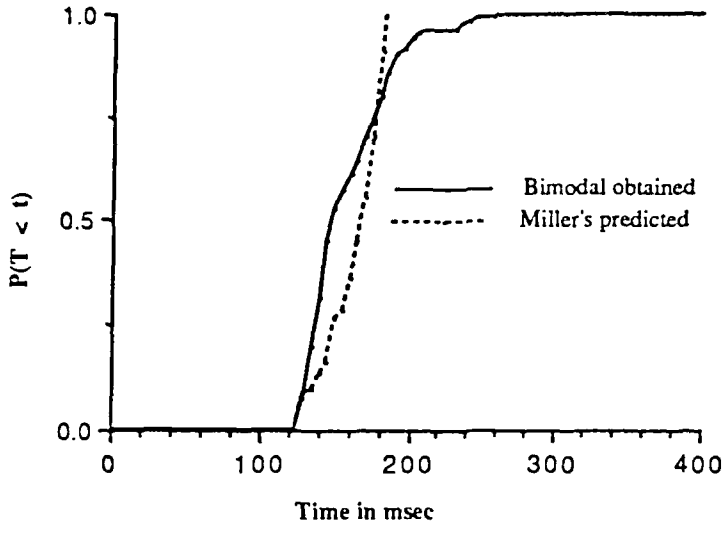
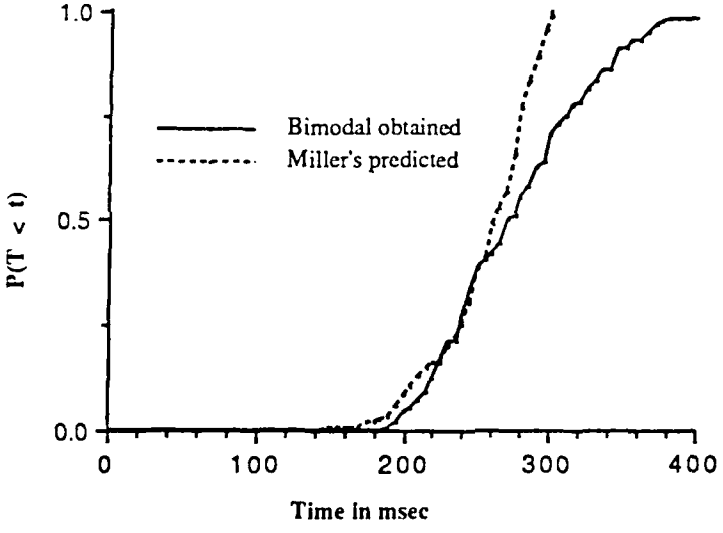

S2: Simple Manual
1.0
0.5
0.0
P(T < t)
0
100
200
300
400
Time in msec
Bimodal obtained
Miller's predicted

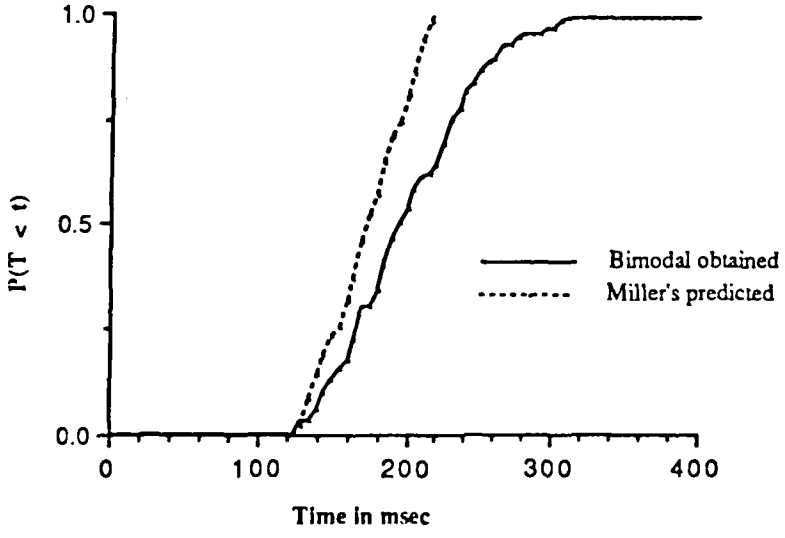
FIGURE 6c S3: Saccades



S3: Directed Manual



S3: Simple Manual



the role of spatial correspondence of the visual and auditory inputs and should therefore substantially clarify this issue.

The evidence for neural summation in the directed manual response task is very weak in comparison to that obtained for saccades, and provides only minimal support for the importance of visual and auditory convergence onto neural elements involved in the execution of this task. The functional organization of the colliculus makes it unlikely that it plays an important role in the initiation of directed manual responses, although similar kinds of mechanisms might exist to control manual responses that are guided by the location of either visual or auditory targets (e.g., polymodal association cortex of the parietal lobe; cf. Anderson, 1987). As in the case of saccades, a reasonable working hypothesis is that bimodal convergence within pathways that are largely sensory in nature are likely to depend upon the spatial alignment of the inputs. Thus, the importance of examining the effects of spatial alignment of the visual and auditory inputs is again suggested, and we intend to investigate this issue concurrently using each of our three model response systems.

In summary, we present evidence that the degree of intersensory facilitation produced by spatially coincident visual and auditory targets varies with the response requirements of the task. In the case of saccades, the facilitatory effects are commensurate with the notion of neural summation of the auditory and visual channels somewhere within the oculomotor pathway. The superior colliculus appears to represent a good candidate structure in the case of intersensory facilitation of saccades. The issue with respect to directed manual responses is less clear. It is noteworthy that an examination of the means displayed in Fig. 4 suggested a substantial degree of facilitation for both saccades and directed-manual responses. However, the S-Measure (Nozawa, Appendix III; Fig 5a-c) clearly indicates that a substantial amount of neural summation was only found for saccades. The effects of presenting visual and auditory targets that are out of spatial register should be quite informative and therefore are of high priority in our future work.

Finally, the neuroanatomical location of the site of this bimodal convergence is of obvious importance. The later phases of this project are intended to examine the effects described in this report in a set of carefully selected neurological patients (described in our original proposal). Careful psychophysical studies of sensory-motor performance in such patients, in conjunction with a detailed neuroanatomical analysis of lesion site (made possible by the brain printing methodology described in Appendix II of this report; Jouandet et al, 1990; Thomas et al., 1990; Tramo et al., 1990), has great potential for providing important information concerning the brain regions that actually support these forms of interactions between sensory modalities.

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APPENDIX I

The Reduction of Saccadic Latency by Prior Offset of the Fixation Point: An Analysis of the "Gap Effect"

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and

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Abstract

The latency to initiate a saccade (saccadic reaction time) to an eccentric target is reduced by extinguishing the fixation stimulus prior to the target onset. Various accounts have attributed this latency reduction (referred to as the "gap effect") to facilitated sensory processing, oculomotor readiness or attentional processes. Two experiments explored the relative contributions of these factors to the gap effect. Experiment 1 demonstrates that the reduction in saccadic reaction time (RT) produced by fixation point offset is additive with the effect of target luminance. Experiment 2 indicates that the gap effect is specific for saccades directed towards a peripheral target and does not influence saccades directed away from the target (i.e. anti-saccades) or choice manual RT. The results are consistent with an interpretation of the gap effect in terms of facilitated premotor processing in the superior colliculus.

The Reduction of Saccadic Latency by Fixation Point Offset: An Analysis of the "Gap Effect"

The latency to initiate a saccade in response to an eccentric target is typically on the order of 180-250 ms (Carpenter, 1977; Wheeless, Boynton & Cohen, 1966). Saccadic reaction times (RT) can be substantially reduced, however, by simply extinguishing the fixation stimulus 200-300 ms prior to target onset (e.g. Fischer & Ramsperger, 1984; Saslow, 1967). In addition to reducing the average latency of saccades, a temporal gap between fixation point offset and target onset (referred to as the "gap condition") may produce a subpopulation of saccades with a modal latency of 120 ms (Fischer & Ramsperger, 1984, 1986). These have been called "express saccades" (e.g., Fischer & Boch, 1983; Fischer & Breitmeyer, 1987; Fischer, 1987). The latency reduction produced by fixation stimulus offsets have been variously attributed to facilitated sensory processing (e.g. Reulen, 1984a), oculomotor readiness (Kalesnykas & Hallett, 1987; Saslow, 1967) or to attentional factors (e.g., Fischer,1987). The present experiments examined the effects of target luminance and response requirements in an attempt to clarify the basis of latency facilitation in the gap condition.

Fixation point offsets could conceivably exert their effect by altering visual sensitivity. It might be easier, for instance, to detect eccentric flashes in a blank field than in the presence of a fixation stimulus. This possibility is considered in Reulen's model (1984a; 1984b) which attributes latency reduction in the gap condition to enhanced processing of the visual target. This model assumes that saccadic RT represents the linear sum of several serially organized processing stages. Following Grice's random threshold theory of response latency (Grice 1968), the model assumes a "sensory stage" in which neural responses to signal onset accumulate until a threshold is reached. Subsequent events represent oculomotor programming

and efferent processes. In this model the accumulation rate is a direct function of signal intensity and is constant over time.

Figure 1 about here

Reulen's model accounts for the reduction in latency in the gap condition by hypothesizing that fixation point offsets increase the rate at which the sensory activity accumulates. The main features of Reulen's model are portrayed in Figure 1 which depicts the accumulation of sensory activity over time for bright and dim flashes. Given a constant threshold, it is evident that bright targets will reach threshold faster than dim ones. This model also predicts that the latency difference between gap and overlap conditions should increase with decreasing target luminance. We tested this possibility directly in the first experiment by measuring saccadic RT to high and low-luminance targets presented with and without a central fixation stimulus.

EXPERIMENT 1

Method

Subjects. The subjects were eight Dartmouth undergraduates and two of the authors (PRL and HCH). All subjects had normal vision or were corrected by contact lenses.

Apparatus. Three computer-controlled red light emitting diodes (LED's) served as the saccade targets and the fixation stimulus. At the viewing distance of 57 cm. each LED subtended 0.5° visual angle. The target LED's were positioned 7.0° to the right or left of the fixation light. The luminance of the fixation light was 0.8 cd/m^2. Target luminance was either 40 cd/m^2 (bright targets) or 0.4 cd/m^2 (dim targets). Luminance was controlled by varying the voltages applied to each lamp via digital to analog converters. The position of the left eye was monitored using the Eye-trac 200 infra-red scleral reflection device which has a resolution

of .25° and a 0-250 Hz. bandwidth. The eye position signal was digitized at a sampling rate of 250 Hz. Head movements were minimized by a chinrest/head restraining assembly.

Design and Procedure. This experiment compared the effects of two levels of target luminance on saccadic RT under the gap and overlap conditions. In the overlap condition the fixation light was illuminated at the start and remained present throughout the trial. In the gap condition the fixation point was illuminated at the start of the trial but was extinguished 200 ms prior to the onset of the eccentric saccade target. Previous work has indicated that optimal gap effects can be obtained with a 200 ms gap interval (e.g. Fischer & Breitmeyer, 1987; Saslow, 1967).

The procedure used in this and the following experiment included several features designed to equate response readiness and minimize anticipatory responses. In previous investigations, fixation point offsets may have alerted the observer to the imminent occurrence of the visual target, perhaps resulting in faster responses than in the overlap condition in which no warning event occurred. In an effort to equate warning cues in the gap and overlap conditions, an auditory warning tone (1000 Hz.) preceded the target on *all* trials. The warning tone was presented via a speaker positioned directly below the fixation LED. To minimize anticipatory responses, the positions of the target were unpredictable, and both fixation conditions included catch trials (20%) in which no target was presented. Gap and overlap trials occurred randomly and with equal frequency within each block of 76 trials.

Figure 2 about here

A schematic illustration of the events in each trial is presented in Figure 2. Each trial began with the illumination of the fixation light. After 900 ms, the 100 ms warning tone was presented. In the gap condition, the fixation light was extinguished simultaneously with the offset of the warning tone, and following a 200 ms dark interval either the right or left LED was illuminated for 300 ms. In the overlap condition, the target was presented 200 ms after the

offset of the warning tone, but the fixation light remained on until 500 ms after target offset. Bright and dim targets were presented randomly within a trial block and occurred equally often in gap and overlap trials. The subjects were instructed to maintain central fixation until the onset of the eccentric target, at which point they were to look at the target as rapidly as possible.

Each subject participated in at least two practice blocks of 76 trials each, followed by six experimental blocks run over a five day period. A maximum of two trial blocks were run in each experimental session.

Data Analysis. Saccades were detected automatically using a velocity criterion (>80 deg sec^{-1}), but were verified by the experimenters. The temporal interval between the onset of the target and the beginning of the saccade was taken as the saccadic latency. Following previous studies (e.g. Fischer & Rampsberger, 1984, 1986), latencies less than 80 ms were counted as anticipations. Latencies longer than 700 ms were considered misses and excluded from the analyses. Saccade magnitudes and peak velocities were computed using calibration data obtained immediately prior to each session.

Results

The latency data for bright and dim targets in the gap and overlap conditions are plotted in Figure 3. A two-way analysis of variance (ANOVA) on these data indicated significant main effects of fixation condition [$F(1,9)=73.2$; $p<.0001$] and intensity [$F(1,9)=472.1$; $p<.0001$], but no significant interaction [$F(1,9)=0.72$]. These results indicate a significant reduction of saccade latencies in the gap condition, and demonstrate that this effect is additive with the facilitatory effect of target luminance. Together, fixation condition and target intensity accounted for 77% of the variance in the data set.

Figure 3 about here

Response Distributions. Since previous studies using the gap paradigm have indicated that express saccades are often revealed as a prominent early mode in latency histograms, we obtained latency histograms for each subject in all conditions. The latency histograms for two representative subjects are shown in Figure 4. As is the case for all subjects, little evidence of bimodality can be discerned by visual inspection of these histograms. Given the differences in procedure used to collect the present data (i.e., the presence of catch trials, inclusion of an auditory warning signal, position uncertainty), we tentatively attribute the absence of a bimodal latency distribution to procedural differences between this and previous work.

Figure 4 about here

Anticipatory responses. While anticipatory responses (i.e. latencies less than 80 ms) were rare, the mean incidence of anticipations was virtually identical in the overlap and gap conditions (1.8% and 1.2%, respectively). The similarity in these anticipation rates argues against the possibility that facilitation in the gap condition is due to a greater tendency to initiate responses prior to the target onset (Kalesnykas & Hallett, 1987).

Saccade Topology. Previous reports describing express saccades have provided little information concerning the topological aspects of saccades in the gap paradigm. We observed some variability in the amplitude (i.e. the size of the saccade expressed in degrees) and velocity of saccades to the targets at 7.0° and therefore analyzed these characteristics. An analysis of the amplitude data indicates only that saccades to the dim targets were slightly but significantly smaller than those to bright targets in both the gap and overlap conditions ($F(1,9)=16.4$; $p=.003$). The average size of the saccades to dim targets at 7.0° was 6.3° versus 6.6° in response to bright targets at the same eccentricity. An analysis of the peak velocities indicated no effect of luminance ($F(1,9)=0.6$) or fixation condition ($F(1,9)=0.4$) .

Discussion

The bimodal latency distributions previously observed in the gap condition (e.g. Fischer & Ramsperger, 1984; Reulen, 1984b) were not found in the present experiment. These data suggest that bimodal distributions are not a necessary correlate of latency reduction in the gap paradigm. In the bright target condition, we observed many saccades with latencies within the range (100-120 ms) in which the subpopulation of "express saccades" has been previously observed. However, none of our subjects showed evidence of a bimodal distribution even though every one demonstrated a reduction in mean RT in the gap condition. Furthermore, the present results suggest that the facilitatory effects of fixation point offset do not depend on the absolute latency of the saccadic responses, since the magnitude of the gap effect was identical for bright and dim targets even though responses to dim targets were approximately 70 ms slower. In addition, analyses of the topology of saccades generated in the gap condition indicate that they do not differ from "regular" saccades with respect to their accuracy or velocity characteristics.

The major finding of this experiment is that the reduction in saccadic RT produced by extinguishing the fixation light is equivalent for bright and dim targets. This addivity between the effects of luminance and fixation condition is incompatible with the mechanisms proposed by Reulen to account for the gap effect. By the logic of additive factors this additivity raises the possibility that target luminance and fixation offsets exert their effects on different processing stages (Sternberg, 1969). Since the time course of neural activity is strongly dependent on stimulus intensity from the level of the photoreceptors to the primary visual cortex (e.g. Baylor & Hodgkin,1973; Lennie, 1983; Miller & Glickstein, 1967), the present findings suggest that fixation point offsets might influence processes subsequent to the loci of intensity dependent effects.

While additive factors logic has generated a coherent and internally consistent account of simple sensorimotor tasks (e.g. Sanders, 1977), it assumes serial, independent processing

stages. This assumption has been challenged in some contexts, however (e.g. Eriksen & Schultz, 1979; McClelland, 1979; Taylor, 1977). Thus one goal of the second experiment was to test further the role of enhanced early sensory processing in mediating the gap effect.

EXPERIMENT 2

If fixation offsets produce enhanced sensory processing in the primary visual pathway, then a variety of responses might show reduced latencies in the gap condition. However, if the effect is restricted to saccades, it would suggest that fixation offsets specifically relate to processes within the oculomotor system. This possibility was tested in Experiment 2, which compared the effects of fixation point offsets on manual RTs and two different types of oculomotor responses: 1) pro-saccades, in which the saccade is directed to the target and 2) anti-saccades, in which the saccades are directed away from the target (Hallett, 1978). In addition, by comparing pro- and anti-saccades, we can evaluate whether the gap effect is a general characteristic of saccadic responses, a possibility suggested by several accounts of latency reduction in the gap paradigm (Fischer & Breitmeyer, 1987; Kalesnykas & Hallett, 1987; Saslow, 1967).

The account originally proposed by Saslow (1967) attributes the reduction of saccade latencies in the gap condition to the saccadic refractory period. Normal saccades and microsaccades are followed by a refractory period lasting approximately 150 ms, during which time a second saccade cannot be initiated (Nachmias, 1959; Carpenter, 1977). Saslow reasoned that subjects are more likely to make micro-corrective saccades when a fixation point is present versus when it is absent. Thus in the overlap condition there is a greater likelihood that subjects are in the midst of the refractory period at the moment of target onset, which delays the saccadic response. According to this view, we might expect both pro- and anti-saccades to be influenced by fixation point offsets, since the saccadic refractory period should affect both types of saccades equally. However, since this refractory period should not influence manual RTs, no effect of fixation offset would be expected in this condition.

More recently, Kalesnykas and Hallett (1987) have proposed that in the gap condition saccades may be programmed prior to the onset of the eccentric target. This view assumes that

saccades are programmed without information about target location, so the parameters of the motor program are initially set in the absence of visual input. According to this hypothesis, anticipatory saccades are initiated too far in advance of the target onset to be modified by its visual coordinates and therefore may be incorrect in direction and/or amplitude. Express saccades are thought to be preprogrammed in the appropriate direction by chance and are initiated late enough to have their amplitude modified by the target coordinates, resulting in short latencies and an accurate movement. Regular latency saccades are those which are either not preprogrammed or were *reprogrammed* in response to the target coordinates. Given that manual movements could also be preprogrammed, this view does not exclude the possibility that fixation offset could reduce choice-manual RTs. It does, however, predict that anti-saccades should show a gap effect, since fixation offsets could trigger that proportion of preprogrammed saccades which happened to be in the direction opposite to the target.

Finally, Experiment 2 has relevance to the proposal that attentional disengagement mediates saccadic facilitation in the gap condition (Fischer, 1987; Fischer & Breitmeyer, 1987). According to this view, extinguishing the fixation point disengages attention. Thus, at the time of target onset, attention is in the disengaged state thereby reducing saccadic latencies by the amount of time normally required to execute the disengage operation. If attention must be disengaged prior to the occurrence of *any* saccade, as Fischer & Breitmeyer (1987) suggest , the latency of anti-saccades might also be facilitated in the gap condition.

Method

Stimuli and Procedure. The stimulus display differed from Experiment 1 in that only the bright target (40 cd/m^2) was used. In addition, a 300 ms gap interval was included and randomly intermixed with 200 ms gap and overlap trials. On both gap and overlap trials, the 100 ms warning tone offset either 200 or 300 ms prior to target onset and coincided with fixation point offset on gap trials. This means that, on overlap trials, there was either a 200 or 300 ms foreperiod between tone offset and target onset. On gap trials this foreperiod

corresponded to the 200 or 300 ms gap duration. In all other respects the stimulus conditions were identical to Experiment 1.

Each subject participated in six trials blocks for each of three response conditions: pro-saccade, anti-saccade and choice-manual RT. Two blocks of 76 trials each were run in each of 9 experimental sessions carried out on separate days. A fixed order of response conditions was used with all subjects participating in the pro-saccade condition first, followed by the anti-saccade condition and finally by the choice-manual RT task. In the anti-saccade task subjects were instructed to saccade the same distance but in the direction opposite the target light. In the choice-manual RT task the subject pressed one of two response keys depending on the location of the target. The stimulus-response mapping was always compatible (e.g., left light - left hand, right light - right hand). At least two blocks of practice trials preceded data collection for each response type. Manual RT's were accurate to the nearest millisecond.

Subjects. Three paid volunteers and two of the authors (PRL and HCH) participated in this experiment.

Results

The latency data are displayed in Figure 5. It is apparent that a strong gap effect emerged only for pro-saccades. This was confirmed statistically in a three-way ANOVA with foreperiod (200 vs. 300 ms), fixation condition (gap vs. overlap), and response condition (manual, pro-saccade, anti-saccade) as repeated factors. In addition to main effects of response condition [$F(2,8)=16.4$; $p<.002$] and fixation condition [$F(1,4)=23.4$; $p<.01$] , this analysis revealed a significant interaction between these factors [$F(2,8)=7.8$; $p<.02$]. Post-hoc comparisons using the Newman-Keuls procedure indicated that the interaction was due to a significant latency reduction produced by fixation offset only for the pro-saccade condition ($p<.05$).

Figure 5 about here

The only other significant effect was an interaction of foreperiod and response type [$F(2,8)=9.5$; $p<.01$]. The means indicate that, regardless of fixation condition, the 200 ms foreperiod was associated with faster responses for both pro- and anti-saccades whereas the 300 ms foreperiod produced slightly faster manual responses. Post hoc analyses using the Newman-Keuls procedure indicate that the difference between foreperiods was significant only for anti-saccades ($p<.05$). The reasons for this foreperiod effect are not clear, but the pattern suggests differences in the time course of alerting processes for different response systems (Ross & Ross, 1980, 1981). In the gap condition, the two levels of foreperiod correspond to gap durations of 200 and 300 ms. The absence of an interaction between foreperiod and fixation condition indicates that gap duration had no reliable effect on RT.

Response Distributions. Latency histograms from two observers for the pro-saccade condition are presented in Figure 6. As in the first experiment, the distributions showed little evidence for bimodality, REP being the lone exception.

Figure 6 about here

Direction Errors and Anticipations. Although generally rare (less than 3% of all responses), direction errors were more common in the anti-saccade condition than in either the pro-saccade or the choice-manual condition. Since only two errors occurred in the manual condition, these data were not analyzed further. The proportion of direction errors occurring in the gap and overlap conditions for pro- and anti-saccades were transformed (arc-sine) and submitted to an ANOVA. This analysis indicated that direction errors were more frequent in the anti-saccade task than in the pro-saccade task [$F(1,4)=39.8$; $p=.004$] and were also more frequent in the gap than in the overlap condition [$F(1,4)=14.5$; $p<.02$]. There was no interaction between these two variables.

The higher frequency of direction errors in the gap condition, again, raises the possibility that the shorter latency in this condition reflects a greater incidence of anticipatory responses. However, the incidence of direction errors was greater for anti-saccades than for pro-saccades and there was no gap effect for anti-saccades. Thus, the facilitatory effects of the gap cannot be explained by a greater tendency to make anticipatory responses. In addition, the frequency of anticipatory responses was found to be unaffected by fixation condition. In the pro- and anti-saccade conditions, 1.7% of all responses had latencies less than 80 ms, whereas none of the manual responses were anticipatory. Proportions of anticipatory responses were computed for each subject, arc-sine transformed and analyzed using a three-way ANOVA with fixation condition, foreperiod and response type (pro- versus anti-saccade) as factors. No main effects or interactions approached significance.

Multiple Saccades. A further analysis of the anti-saccade data revealed a strong tendency for four of the subjects to generate "double saccades". For these subjects 38% of the responses in this condition were characterized by primary and secondary saccades. In general the magnitude of the secondary saccade was larger (greater than 60% of the primary) than is typical of corrective saccades (Carpenter, 1977). In his investigations of the anti-saccade paradigm, Hallett (1978; Hallett & Adams, 1980) also observed many multiple saccades in this task. Interestingly, Jay and Sparks (1990) report many double saccades in response to acoustic targets, suggesting that such saccadic responses may be a characteristic of saccades executed without visual guidance. Finally, it should be noted that the presence or absence of the fixation point had no effect on the frequency of double saccades.

General Discussion

Efferent Factors in the Gap Paradigm

The results from Experiment 2 indicate that the facilitatory effect of fixation stimulus offset is limited to pro-saccades. This response-specificity, together with the finding that the gap effect is additive with the effect of target luminance, appears inconsistent with views that

attribute the facilitatory effects of fixation point offsets to enhanced early visual processing. Both the additivity with target luminance and the response specificity are, however, consistent with the hypothesis that the gap effect may be related to pre-motor processes specifically within the oculomotor system.

While the accounts of the gap effect offered by Saslow (1967) and Kalesnykas and Hallett (1987) also emphasize efferent factors, aspects of the present results are not readily explained by either view. If saccade direction and amplitude are preprogrammed in the gap condition or if the saccadic refractory period contributes to the gap effect, we would expect anti-saccade latencies to be facilitated by fixation point offset. The results from Experiment 2 provide no support for these expectations.

Other aspects of the data are inconsistent with the preprogramming hypothesis offered by Kalesnykas and Hallett (1987). By their view, the tendency to initiate preprogrammed saccades in the gap condition should produce more anticipatory saccades relative to the overlap condition. Using 80 ms as a criterion for anticipations, we did not find this to be the case. Kalesnykas and Hallett (1987) propose that since anticipations tend to be hypometric, saccade amplitude should also be used to distinguish these responses from express saccades. We found no differences in saccade amplitude in the gap and overlap conditions, indicating that our data set does not include substantial numbers of hypometric anticipatory responses.

Anticipatory factors may have played a greater role in some earlier work on the gap paradigm which did not use catch trials, unpredictable target locations or provide warning signals in both the gap and overlap trials. In many previous studies (e.g. Fischer & Rampsberger, 1986; Mayfrank et al., 1986; Reulen, 1984a,1984b), there is good reason to suggest that, since the fixation point offset provided the only warning of the impending target event subjects were more alert and prepared to respond in the gap condition than in the overlap condition (c.f. Ross and Ross, 1980, 1981). The present data were obtained under conditions specifically designed to minimize the contribution of anticipatory processes. These measures

appear to have succeeded in minimizing anticipatory effects, as evidenced by the similarity in anticipatory errors in the gap and overlap conditions.

Role of the Superior Colliculus in Saccadic Facilitation

Schiller, Sandell and Maunsell (1987) report that ablations of the superior colliculus (SC) abolish express saccades, while producing only a modest slowing of regular saccades. The close coupling of the sensory and motor fields in the SC and its direct output to brainstem oculomotor centers, make it well-suited for controlling the rapid foveation of eccentric targets (Sparks & Mays, 1980; Wurtz & Goldberg, 1972). Since observers are not normally compelled to fixate every peripheral event, some form of inhibitory control must be exerted on collicular mechanisms. Hikosaka and Wurtz (1983) have recently established a neural basis for such inhibition by demonstrating that activity in substantia nigra tonically inhibits the SC. Furthermore, electrophysiological observations indicate that the threshold current needed to elicit a saccade from either the SC or the frontal eye field (FEF) increases during active foveation (Goldberg, Bushnell & Bruce, 1986). Thus it seems reasonable to suggest that the reduction in latency observed in the gap condition may reflect the functioning of collicular mechanisms in the absence of this fixation-related inhibition (i.e. fixation release). The absence of a gap effect for either choice-manual responses or anti-saccades could be attributed to a lack of collicular involvement in either of these responses.

The FEFs have been implicated in the control of anti-saccades since patients with focal excisions involving this region are selectively impaired on this task (Guitton, Buchtel, & Douglas, 1985). Signals originating in the FEF can control eye position independently of the SC (Schiller True & Conway, 1980), perhaps via direct neuroanatomical connections to brainstem oculomotor centers (e.g.Leichnetz, 1981; Leichnetz, Smith & Spencer, 1984). To the extent that the FEF's can directly control saccades, responses requiring the FEF, such as anti-saccades, may be independent of neural influences exerted on the colliculus. We suggest that a possible reason that anti-saccades are not be influenced by the gap condition is because

their generation may be less dependent on the SC where fixation point offsets appear to exert their effects. Given the highly voluntary nature of anti-saccades, it is unlikely that special mechanisms would have evolved to inhibit their occurrence, as is the case for pro-saccades.

The hypothesis that the gap effect reflects facilitated pre-motor processes that are specific to pro-saccades suggests the possibility that the deeper layers of the SC might play an important role in this phenomenon, since many cells in this region are not visually responsive but show pre-saccadic activity (e.g. Jay & Sparks, 1987). It would therefore be interesting to compare the activity of such pre-saccadic burst neurons in the gap and overlap conditions.

Attentional Mediation of Latency Facilitation?

While we take the view that the latency facilitation in the gap condition reflects the release of oculomotor mechanisms from the inhibitory influences engaged during active fixation, Fischer and his colleagues attribute these effects to attentional disengagement (Fischer, 1987; Fischer & Breitmeyer, 1987; Mayfrank et al. 1986). Several features of our data, caution us against invoking attentional mechanisms to explain the gap effect. Considerable electrophysiological and behavioral evidence indicates that attention influences early stages of sensory processing (e.g. Hawkins, Shafto & Richardson, 1988; Mangun, Hillyard & Luck, in press). The work of Hawkins et al. (1988) is particularly relevant to the logic of the present investigation. They found that the magnitude of attentional effects produced by spatial precues interacted with target luminance, leading them to conclude that attention, like signal intensity, affected early visual processing (also see Backus & Sternberg, 1988). Furthermore, as Posner and his colleagues originally demonstrated (Posner, Nissen & Ogden, 1978, Posner, 1980), the facilitatory effects of attentional precues are not restricted to one response modality but can enhance manual as well as saccadic RT. In contrast, the present results suggest that the gap effect operates quite differently from attentional manipulations in precuing paradigms, since it is additive with target luminance and is apparently restricted to pro-saccades. Thus, if attentional mechanisms contribute to the gap effect, they differ from

those mechanisms that mediate stimulus selection in spatial precuing paradigms and may be related instead to the selection of the appropriate oculomotor program (cf. Goldberg & Seagraves, 1987).

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Figure Captions

Figure 1. Diagram of Reulen's facilitation model. In this model a sensory integrator accumulates activity following a peripheral afferent delay which is a function of signal intensity. The rate of accumulation is jointly determined by signal intensity and a facilitation factor produced by fixation point offset. The facilitation factor, per se, is independent of target intensity. In this diagram the parameters representing the facilitation factor and target intensity were multiplicatively combined, following equation 1 of Reulen (1984a). Note that the time difference to reach threshold in the gap (G) versus the overlap (O) condition is larger for the dim than for the bright target. This interaction between the effects of intensity and fixation condition is predicted by the model regardless of whether the facilitation and intensity parameters are additively or multiplicatively combined.

Figure2. Schematic representation of trial events in Gap and Overlap conditions for Experiments 1 and 2.

Figure 3. Mean saccade latencies to bright and dim targets in the gap and overlap conditions.

Figure 4. Frequency histograms representing the saccade latencies to bright and dim targets in the gap and overlap conditions for two observers (bin width=8).

Figure 5. Mean latencies for the three response modes (pro-saccades, anti-saccades and choice-manual responses) in the gap and overlap conditions.

Figure 6. Frequency histograms representing the pro-saccade latencies for two observers in the gap and overlap conditions with 200 and 300 ms foreperiods (bin width=8).

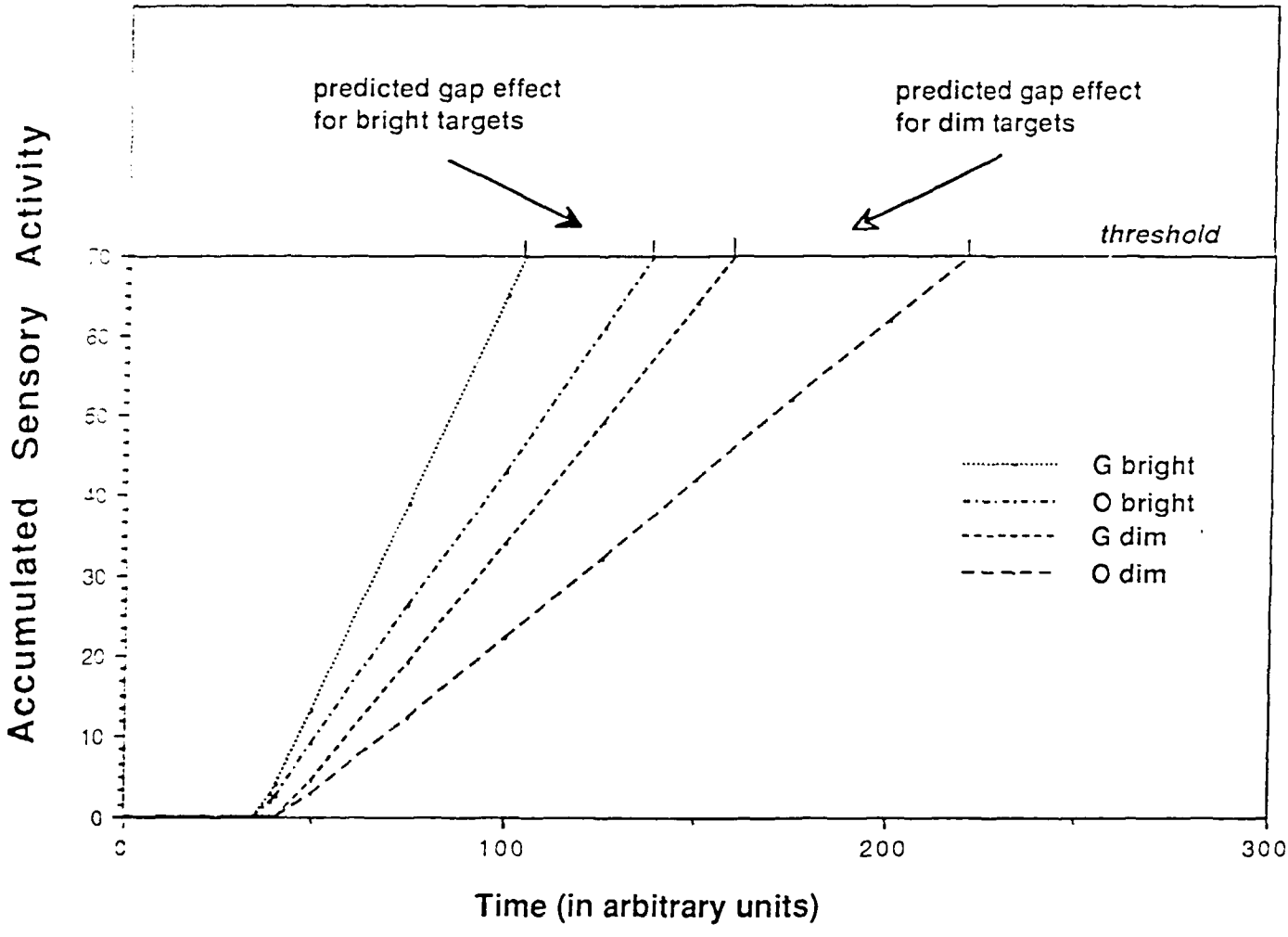
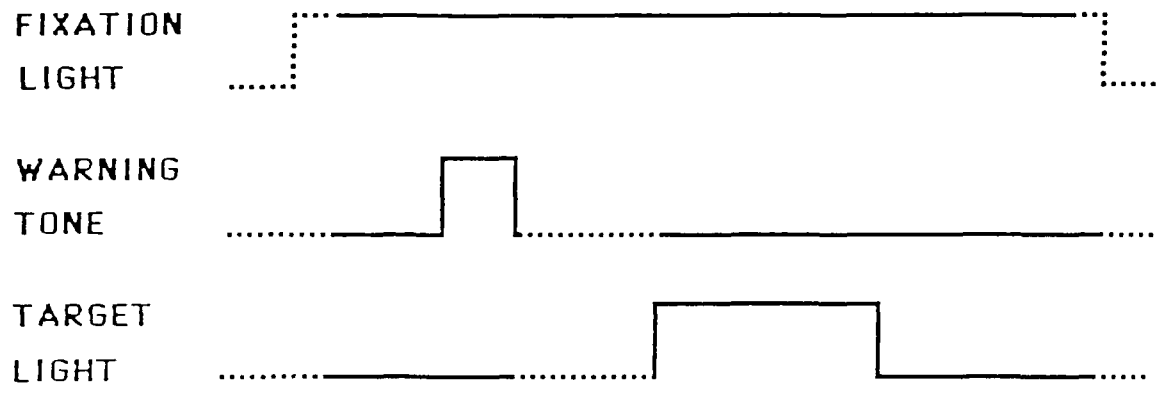
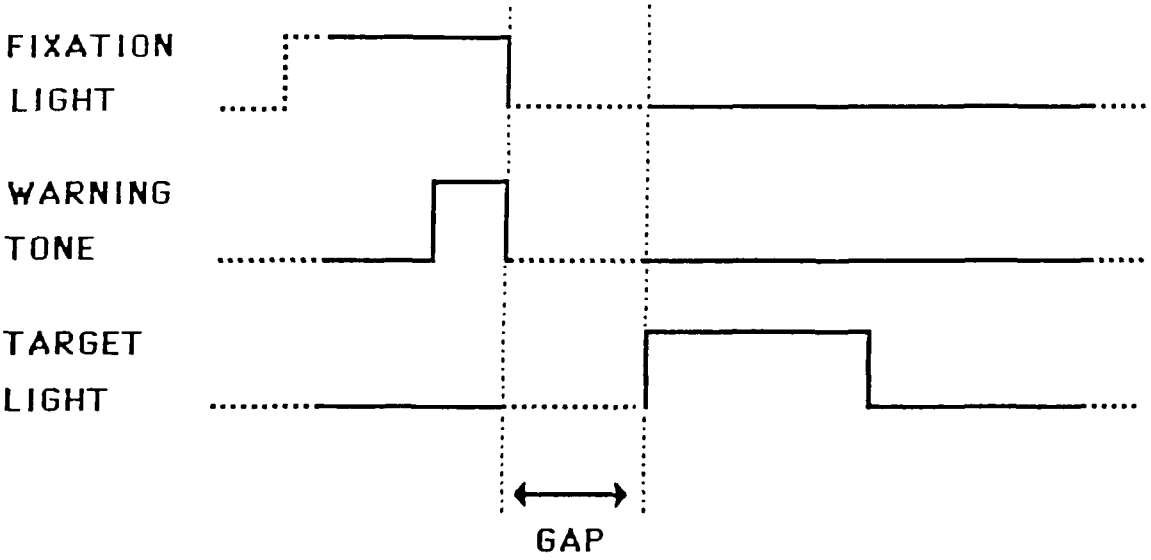

predicted gap effect
for bright targets
predicted gap effect
for dim targets
threshold
G bright
O bright
G dim
O dim
Accumulated Sensory Activity
0
10
20
30
40
50
60
70
0
100
200
300
Time (in arbitrary units)

Figure 1

OVERLAP CONDITION



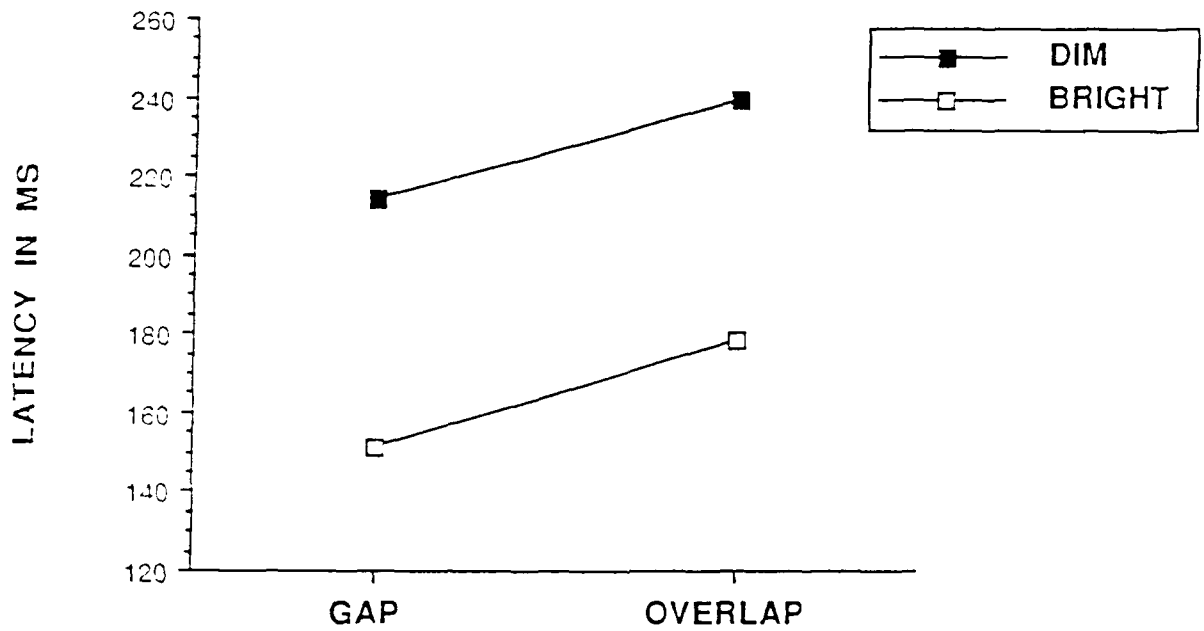
GAP CONDITION



200 ms

(or 300 ms in Experiment 2)

Figure 2


LATENCY IN MS
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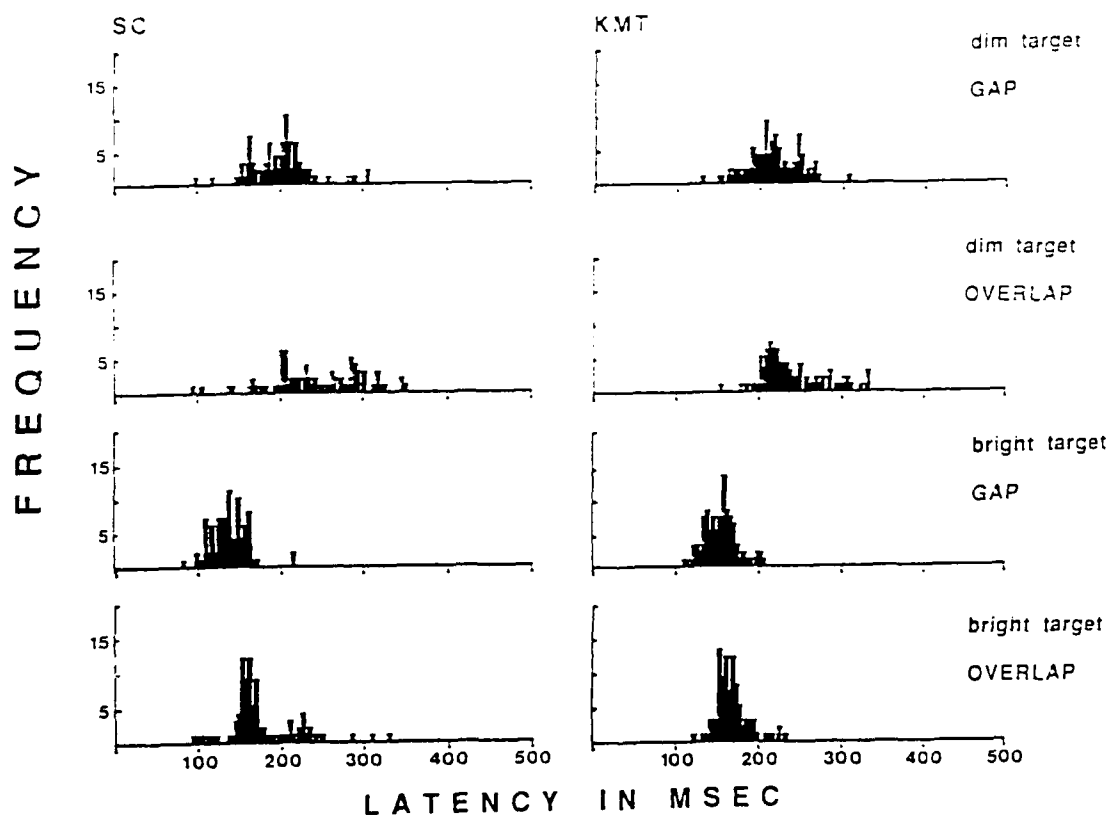
140
120
GAP
OVERLAP
DIM
BRIGHT

Figure 3


SC
KMT
dim target
GAP
dim target
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bright target
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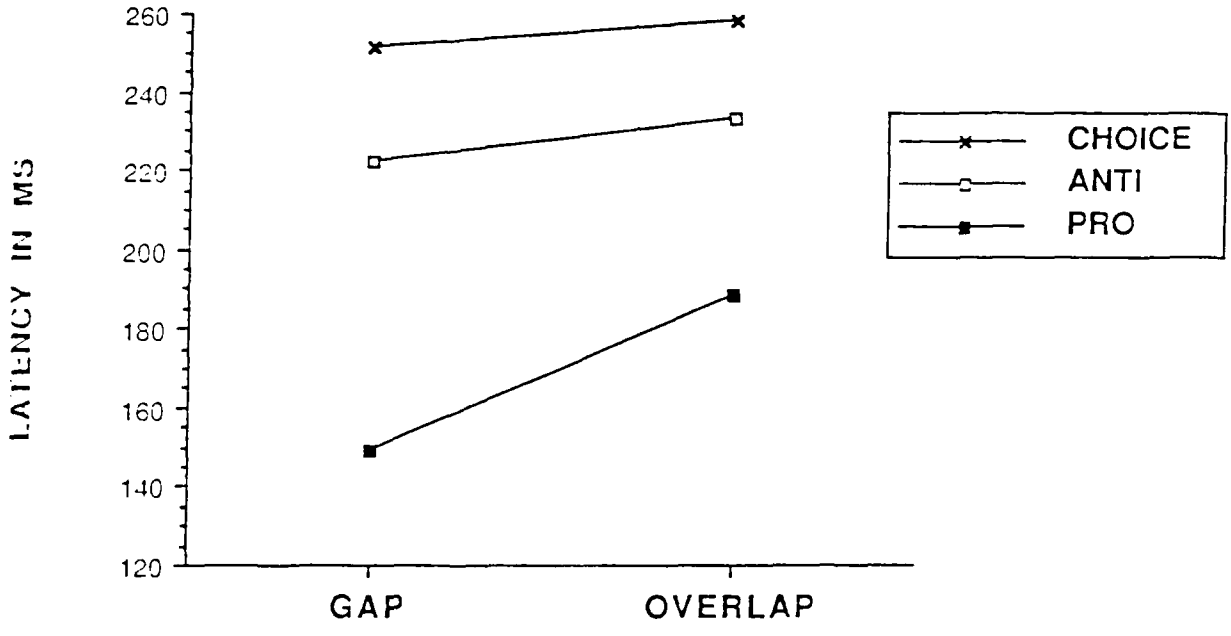
LATENCY IN MSEC

Figure 4


LATENCY IN MS
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CHOICE
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Figure 5

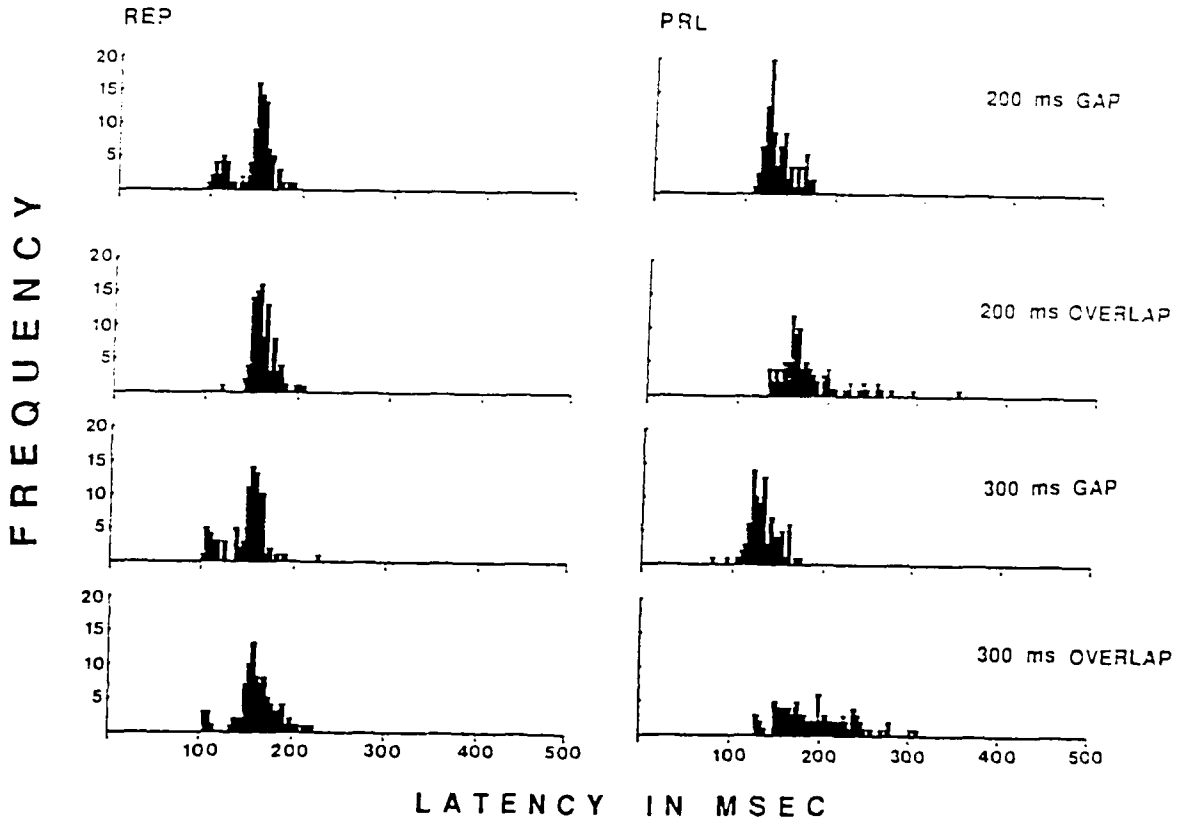
REP
PRL
200 ms GAP
200 ms OVERLAP
300 ms GAP
300 ms OVERLAP
FREQUENCY
LATENCY IN MSEC
100
200
300
400
500

Figure 6

APPENDIX II

Music Perception and Cognition Following Bilateral Lesions of Auditory Cortex

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Abstract

■ We present experimental and anatomical data from a case study of impaired auditory perception following bilateral hemispheric strokes. To consider the cortical representation of sensory, perceptual, and cognitive functions mediating tonal information processing in music, pure tone sensation thresholds, spectral intonation judgments, and the associative priming of spectral intonation judgments by harmonic context were examined, and lesion localization was analyzed quantitatively using straight line two dimensional maps of the cortical surface reconstructed from magnetic resonance images.

Despite normal pure tone sensation thresholds at 250–8000 Hz, the perception of tonal spectra was severely impaired, such that harmonic structures (major triads) were almost uniformly judged to sound dissonant; yet, the associative priming of spectral intonation judgments by harmonic context was preserved, indicating that cognitive representations of tonal hierarchies in music remained intact and accessible. Brainprints demonstrated complete bilateral lesions of the transverse gyri of Heschl and partial lesions of the right and left superior temporal gyri involving 98 and 20% of their surface areas, respectively. In the right hemisphere, there was partial sparing of the planum temporale, temporoparietal junction, and inferior parietal cortex. In the left hemisphere, all of the superior temporal region anterior to the transverse gyrus and parts of the planum temporale, temporoparietal junction, inferior parietal cortex, and insula were spared.

These observations suggest that (1) sensory, perceptual, and cognitive functions mediating tonal information processing in music are neurologically dissociable, (2) complete bilateral lesions of primary auditory cortex combined with partial bilateral lesions of auditory association cortex chronically impair tonal consonance perception, (3) cognitive functions that hierarchically structure pitch information and generate harmonic expectancies during music perception do not rely on the integrity of primary auditory cortex, and (4) musical priming may be mediated by broadly tuned subcomponents of the thalamocortical auditory system. ■

INTRODUCTION

The orderly arrangement of anatomical zones within the auditory cortex has inspired a number of authors to assign fine structure a role in functional specialization. The belief that structural complexity and connectivity patterns reflect functional complexity and hierarchical processing was expressed long ago by Campbell (1905), who designated the granular cortex of the supratemporal plane "audiosensory" and the surrounding belt "audiopsychic." However, empirical data about the organization of cognitive functions mediating auditory information processing outside the verbal domain remain scarce. Although the cortical representation of sensory information (e.g., frequency receptive fields) has been extensively documented in the cat and monkey using neuronal evoked potential techniques (for reviews see Aitkin et al. 1984; Brugge and Reale 1985), we know little about how physical features of acoustic signals are processed to form psychological representations of sound, and how knowledge about sound contributes to sensory integration and percept formation. The effects of bilateral ablations in animals indicate that these higher levels of processing rely on the integrity of auditory cortex (for reviews see Neff et al. 1975; Whitfield 1985); the relative roles of the primary and secondary areas have not yet

been distinguished. In many physiological studies employing depth electrodes (Celesia 1976), magnetoencephalography (Hari et al. 1980; Romani et al. 1982), and radioisotope imaging (Lauter et al. 1985) have primarily focused on the problems of boundary definition and tonotopicity, and have therefore used elementary acoustic stimuli such as clicks and pure tones to evoke cortical responses. Anatomical data derived from the study of neurological populations are likewise wanting given the rarity of auditory nonverbal deficits gross enough to cause overt symptoms following focal unilateral lesions, the rarity of focal bilateral and symmetrical lesions, and the proximity and common vascular supply of the primary and secondary auditory areas. Furthermore, brain scans obtained for clinical indications provide a limited view of the supratemporal plane because of their orientation (horizontal) and slice thickness (8–10 mm), and therefore frequently fail to capture the transverse gyri of Heschl, which typically house the primary auditory areas, and the full extent of the superior temporal gyrus, temporoparietal junction, parietal operculum, and temporal pole, which typically house the auditory association areas (Galaburda and Sanides 1980).

In the present study, we examined a well documented case of cortical hearing loss (Mendez and Geehan 1988) under experimental conditions designed to tap selectively into sensory, perceptual, and cognitive functions mediating tonal information processing. The choice of a musical priming paradigm (Fig. 1; Bharucha and Stoeckig 1986, 1987) reflected the desire to engage cognitive processes that hierarchically structure sensory information and thereby facilitate spectral pattern perception. The cortical mapping procedure of Jouandet et al. (1989) permitted the localization and quantification of damage to the presumed gross anatomical landmarks of the primary auditory cortex and auditory association areas imaged by thin-section coronal magnetic resonance scanning (MR).

RESULTS

Pure Tone Sensation Thresholds

Intensity thresholds for detecting a 500 msec pure tone at 250, 500, 1000, 2000, 4,000, and 8000 Hz were within normal limits for each ear (Fig. 2).

Spectral Intonation Judgments

Intonation judgments about harmonic spectra (major triads) and quasiharmonic spectra (mistuned major triads) were performed better than chance (56%, $t(47) = 2.00$, $p < .05$), but overall accuracy was more than 2 SD below the mean of normals' performance on the more difficult priming task (83 ± 11%, Bharucha and Stoeckig 1987).

Associative Priming of Spectral Intonation Judgments

M.S.'s performance on the musical priming task is illustrated in Figure 3 alongside data previously obtained from 13 normal subjects using the same stimuli and procedure.

Overall accuracy was at chance (48%, $t(95) < 1$) and fell greater than three standard deviations below the mean of the normal population (07th percentile). There

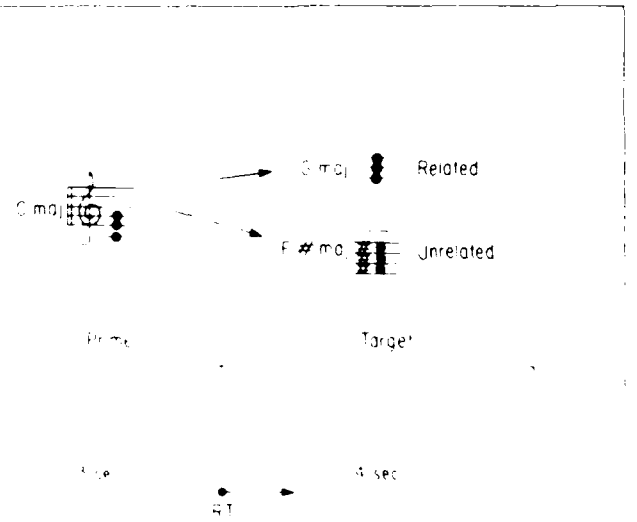
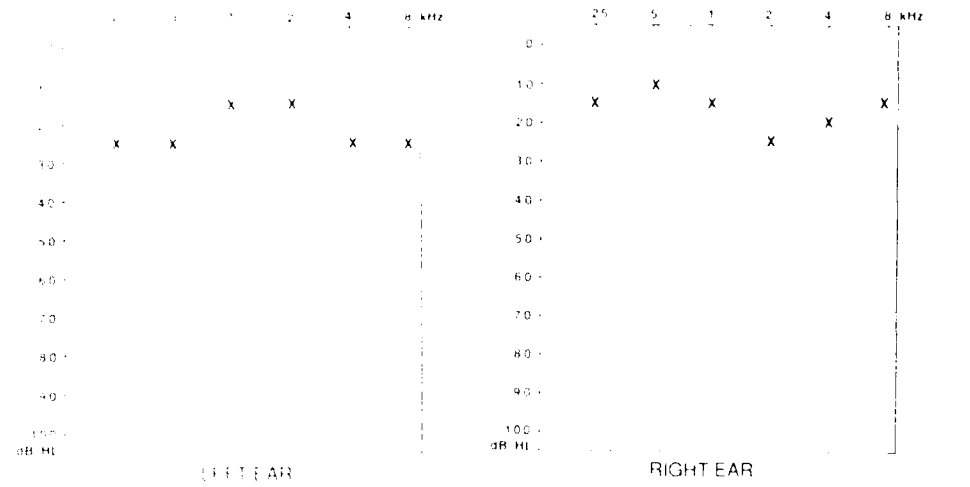


Figure 1.



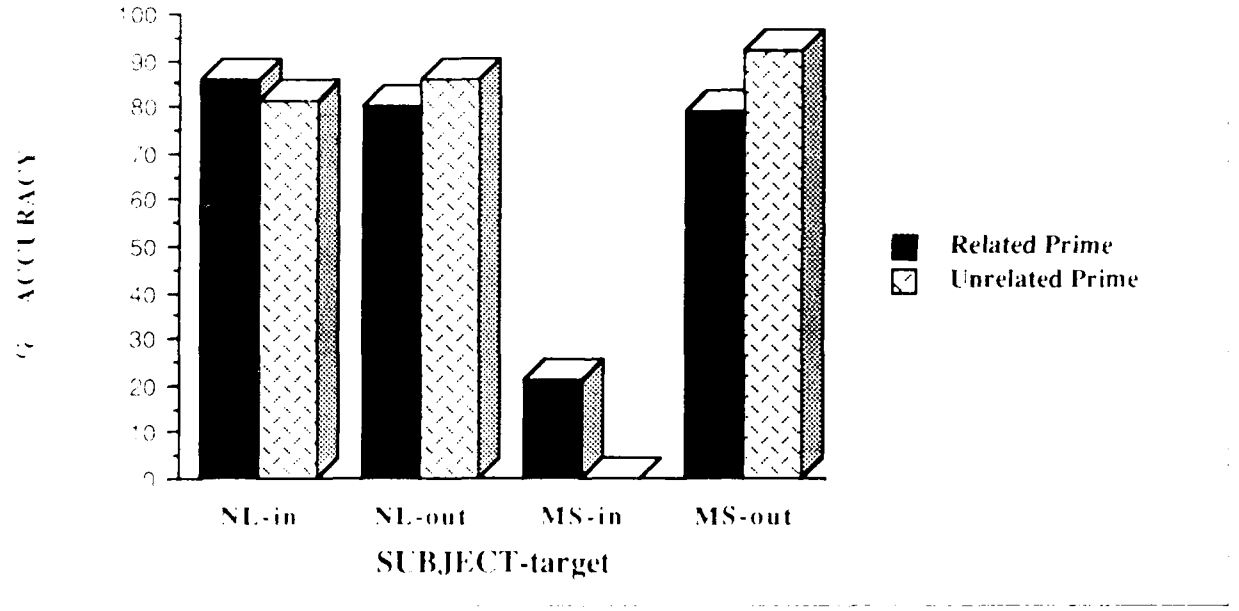


Figure 3. [illegible]

was a marked bias to judge target chords as out of tune ($F(1,28) = 112.06$, $p < .0001$). However, the interaction between target intonation and the harmonic relatedness of the prime to the target was significant in the predicted direction: $F(1,28) = 5.41$, $p < .05$. As in normals, MS was more accurate when the in tune target chords were preceded by a prime chord that was harmonically related to the target. In 12 unstimulated target chords were preceded by a prime chord that was not harmonically related to the target. In none of the 24 in tune unrelated trials was the target correctly perceived to be in tune. Statistical analysis of the intonation relatedness interaction using reaction time data was precluded by the absence of any correct responses in the in tune unrelated condition.

Brainprints and Quantitative Lesion Localization

Stereographic two-dimensional surface maps of the unfolded right and left cerebral hemispheres are illustrated in Figures 4 and 5, respectively. Coronal MR sections through the superior temporal region representative of those used to map the auditory cortex are shown in Figure 6. The surface areas (SA) of individual regions of

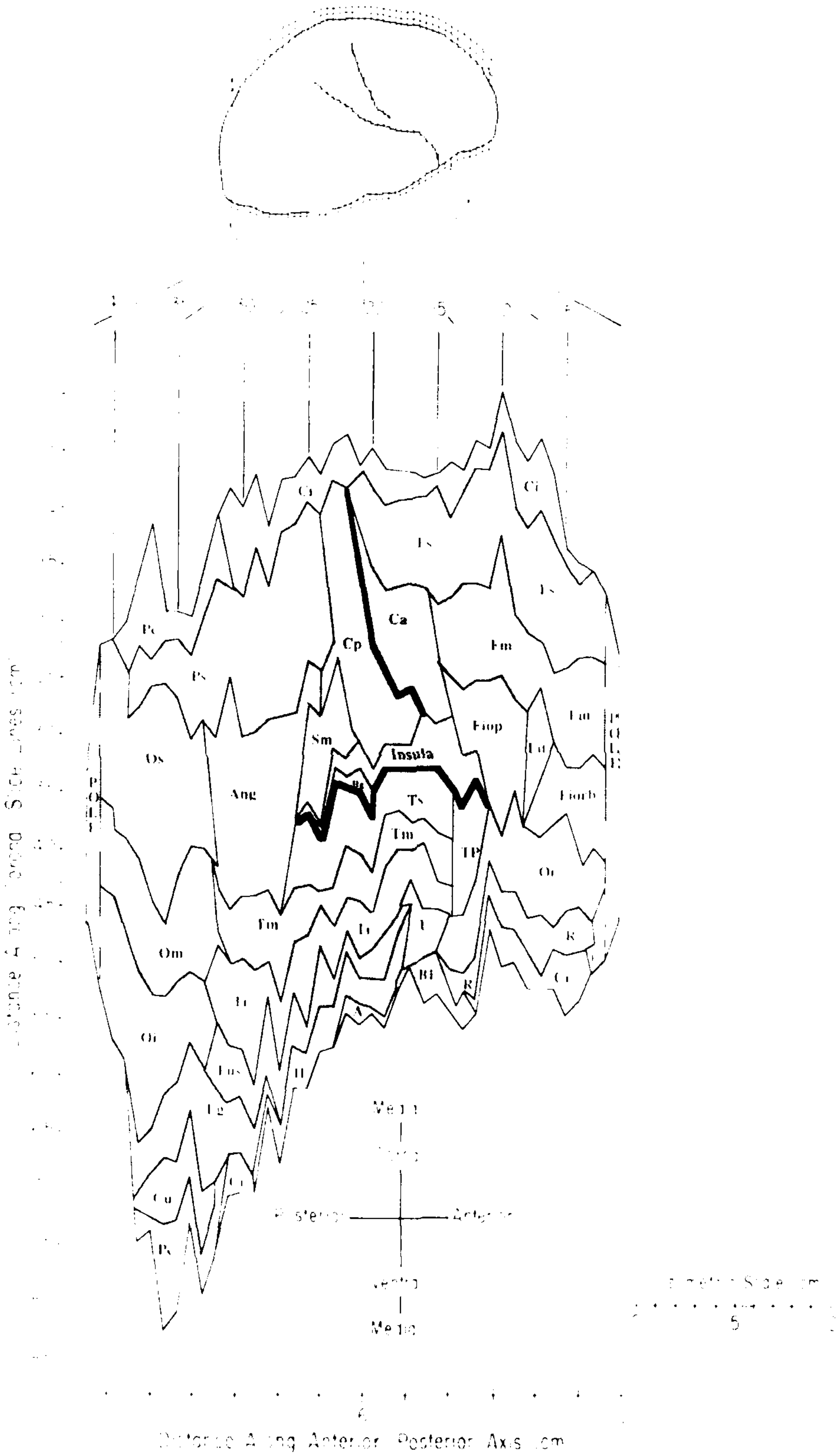


Figure 4. Brain print of M.S.'s right cerebral cortex. Coronal MR sections are numbered sequentially from anterior to posterior (right to left on the page). Gray areas indicate intrasulcal cortex; yellow area indicates cortex lying within and/or overlying abnormal MR signal. Regional surface area measurements are given in Table 1 and regional lesion measurements in Table 2. For glossary of abbreviations, see Appendix.

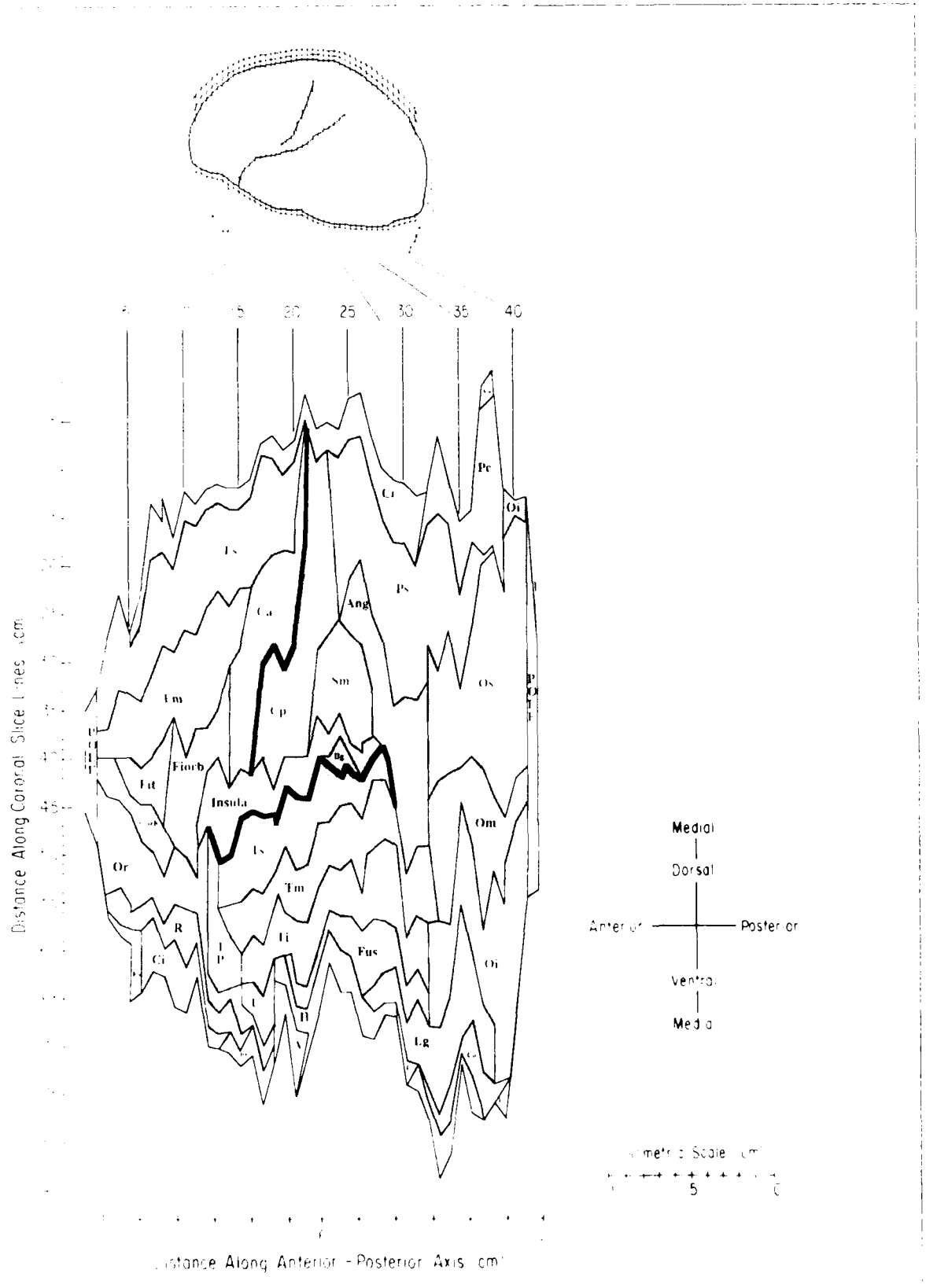


Figure 5. Reconstruction of MRI left hemisphere coronal MR sections are numbered sequentially from anterior to posterior (left to right on the page). See Figure 4 for details of section placement. Tables 1 and 2 for quantitative measures, and the Appendix for abbreviations.

Table 1. Regional Cortical Surface Area Measurements for the Right and Left Hemispheres

	Right Hemisphere		*Left Hemisphere*	
	cm²	*% hemisphere*	*cm²*	*% hemisphere*
Temporal Lobe	159	20	176	20
Transverse gyrus	3		3	
Superior temporal gyrus	27		33	
Temporal pole	9		8	
Middle temporal gyrus	52		43	
Inferior temporal gyrus	40		41	
Hippocampal region	15		11	
Uncus	5		5	
Amygdala	2		2	
Fusiform	26		21	
Parietal Lobe	188	23	208	23
Postcentral gyrus	52		44	
Supramarginal gyrus	12		21	
Angular gyrus	45		55	
Superior parietal lobule	68		72	
Precuneus	81		86	
Frontal Lobe	236	30	247	27
Superior frontal gyrus	74		72	
Middle frontal gyrus	40		45	
Inferior frontal gyrus				
Pars orbitalis	16		10	
Pars triangularis	6		9	
Pars opercularis	22		22	
Precentral gyrus	22		57	
Orbitofrontal gyri	33		38	
Rectal gyrus	17		16	
Occipital Lobe	151	19	190	21
Lateral occipital gyri	113		149	
Cuneus	12		11	
Lingual	26		30	
Other Regions				
Insula	17		28	
Paracingulate area	017		019	
Cingulate gyrus	68		58	
Basal forebrain	7		5	
Total Hemisphere	800		899	

Table 2. Percent Regional Cortical Surface Area Contained within or Undercut by the Lesion

	Right Hemisphere			*Left Hemisphere*		
	% lesion	*% within*	*% undercut*	*% lesion*	*within*	*% undercut*
Temporal Lobe	64	27	37	7	7	—
Transverse gyrus of	100	100		100	100	
Superior temporal gyrus	98	76	22	20	20	—
Temporal pole	100	100		—		—
Middle temporal gyrus	70	43	27	4	4	
Inferior temporal gyrus	53	12	41	—	—	
Hippocampal region	68		68	—		—
Uncus	100	33	67	—	—	—
Amygdala	100		100	—	—	—
Fusiform	35		35		—	—
Parietal Lobe	45	14	31	15	9	6
Postcentral gyrus	91	48	43	—	—	—
Supramarginal gyrus	100	53	47	32	32	—
Angular gyrus	40	2	38	56	33	23
Superior parietal lobule	29	3	26	5	—	5
Precuneus					—	—
Frontal Lobe	40	19	21	—	—	—
Superior frontal gyrus	15		15			
Middle frontal gyrus	48	23	25			
Inferior frontal gyrus						
Pars orbitalis	9		9			
Pars triangularis	59		59			
Pars opercularis	100	88	12			
Precentral gyrus	94	49	45			
Orbital gyri	35	11	24			
Rectal gyrus	5		5			
Occipital Lobe				2	1	1
Lateral occipital gyri				4	2	2
Cuneus					—	
Lingual						
Occipital pole						
Insula						
Insula	100	100		9	9	
Transverse temporal gyri	100	100		100	100	
Cingulate gyrus						
Paracentral lobule	4	4				
Limbic lobe gyri	34	17	19	3	4	1

interest are listed in Table 1 and the proportion of each region that was affected by the lesion is listed in Table 2.

There were complete, bilateral lesions of the transverse gyri of Heschl. Their SA measured 3 cm² on each side, almost 2 SD below the mean of previous normal measurements obtained by the same method (7.6 ± 2.5 cm², N = 32; Tramo et al. 1990 and unpublished data).

In the right hemisphere (Figure 4), almost all superior temporal gyrus SA (98%) and all temporal pole SA were contained within and/or overlay areas of abnormal MR signal. Most of the planum temporale was undercut by the lesion; a small portion of its posterior extension into and including the temporoparietal junction was spared. Elsewhere in the temporal lobe, 70% of middle temporal gyrus SA and 83% of inferior temporal gyrus SA were affected by the lesion. There was extensive involvement of medial temporal structures, including 68% of the hippocampal region, all of the amygdala, and all of the uncus. In the parietal lobe, all of the operculum was involved; 100% of supramarginal gyrus SA, 91% of postcentral gyrus SA, and [illegible] of angular gyrus SA were within or undercut by the lesion. All of the insula was involved. In the frontal lobe, the pars opercularis was completely infarcted; in addition, the posterior portions of the pars orbitalis (58% SA), pars triangularis (59%), middle frontal gyrus (18% SA), superior frontal gyrus ([illegible] SA), and orbitofrontal gyri (35% SA) and almost all of the precentral gyrus (94% SA) were involved. Occipital lobe structures were completely spared.

In the left hemisphere (Figure 5), the posterior 20% of superior temporal gyrus SA, including all but small posterolateral segments of the planum temporale and temporoparietal junction, was contained within areas of abnormal MR signal; the superior temporal gyrus anterior to the transverse gyrus and all of the temporal pole were spared. Four percent of adjacent middle temporal gyrus SA were affected by the lesion, mostly within the superior temporal [illegible]. In the parietal lobe, much of the operculum was [illegible] posteriorly; 32% of supramarginal gyrus SA and [illegible] of angular gyrus SA were involved. The anterior 91% of insula SA was spared. Small portions of the superior parietal lobule ([illegible] SA) and [illegible] lateral occipital gyri ([illegible] SA) were involved.

In the right hemisphere, the lesion extended deep into the [illegible] white matter, putamen, caudate, [illegible] globus pallidus, claustrum, internal capsule, external capsule, and extreme capsule [illegible] comprised [illegible] total right hemisphere volume. The left hemisphere lesion was largely superficial and comprised [illegible] total left hemisphere volume.

DISCUSSION

[illegible]

grossly inaccurate and there was a response bias indicating that tonal consonance perception was severely impaired, and (3) the associative priming of spectral intonation judgments by harmonic context was preserved. Brainprints demonstrated (1) complete bilateral lesions of the transverse gyri of Heschl and (2) partial bilateral lesions of the superior temporal gyri involving 98% of the cortical surface area on the right and 20% on the left. The right hemisphere infarct spared portions of the planum temporale, temporoparietal junction, and inferior parietal cortex; the left hemisphere infarct spared all of the anterior superior temporal gyrus and temporal pole and parts of the planum temporale, temporoparietal junction, inferior parietal cortex, and insula.

Functional Dissociations

The perception of tonal consonance is determined by the frequency relationships among simultaneous elements within tonal spectra (Helmholtz 1863/1954; Levelt et al. 1966; Terhardt 1974, 1984) and is influenced by the harmonic context established by prior spectra (Bharucha and Stoeckig 1986, 1987; Marin and Barnes 1985). Normal subjects exhibit a dissociation between spectral content and associative priming as they influence intonation judgments: degrading the spectrum by removing some harmonics renders the intonation judgment more difficult but does not diminish the priming effect (Bharucha and Stoeckig 1987). In MS, frequency information could be detected at normal thresholds, but fine-grained analysis of the frequency relationships among major triad components was severely impaired. Still, top-down influences on tonal consonance perception remained operative. Sensory, perceptual, and cognitive functions mediating tonal information processing thus appear to be neurologically dissociable.

A number of authors have proposed that auditory percepts are reconstructed from fragments of sound at higher associative levels of processing (for reviews see Hartmann 1988; Whitfield 1985). That associative processes which generate harmonic expectancies in music can be activated even when elementary components of musical stimuli are degraded or the capacity to make discriminations at this level is lost suggests that synthetic processes (e.g., the integration of frequency relationships into a harmonic whole) do not depend entirely upon fine-grained analytic processes (e.g., the ability to resolve frequency differences between individual spectral components in timbre perception).

Lesion Localization

[illegible]

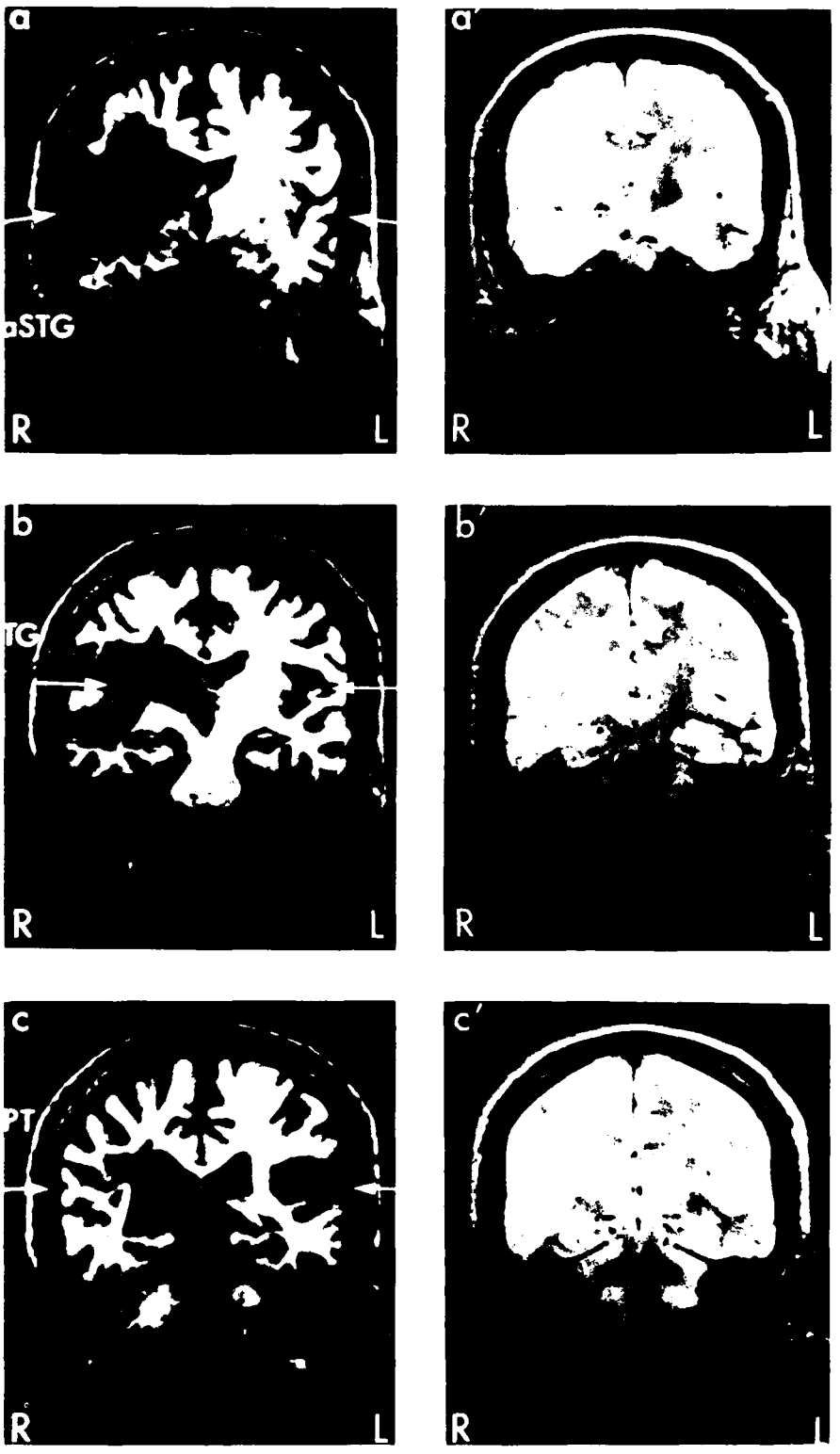


Figure 6. [illegible] MR images at the perisylvian region representative of the 20 sections through the area depicted in the brainprints. MRs a [illegible] were digitized images respectively of the anterior superior temporal gyrus (aSTG). MRs b and b' are sections through the [illegible]. The T2 weighted image clearly demonstrates left as well as right sided hyperintense signal abnormalities [illegible]. PT is that portion of the superior temporal gyrus lying posterior to the transverse gyrus of [illegible] as well as the deep white matter and basal ganglia in the right hemisphere.

complete, bilateral lesions of the koniocortical fields. In the right hemisphere, some of the temporoparietal isocortex and perhaps a small posterior portion of the external parakoniocortex may have been spared. Much of the remaining posterior association belt on the right, including most of the external and all of the internal and caudodorsal parakoniocortex, appears to be undercut by the lesion. Right perisylvian structures anterior and medial to the transverse gyrus, including the rostral parakoniocortex, prokoniocortex, rostral isocortex, parainsular zone, and temporopolar proisocortex, appear to be infarcted. In the left hemisphere, small posterolateral portions of temporoparietal isocortex and perhaps external parakoniocortex may have been spared; much or all of the rostral parakoniocortex, isocortex, and proisocortex along the anterior superior temporal gyrus, temporal pole, and parainsular zone appear to be spared. All of the internal, external, and caudodorsal parakoniocortex as well as the prokoniocortex on the left may have been infarcted.

The physiological properties of neurons within the perisylvian areas that are spared in M.S. can only tentatively be surmised on the basis of their response characteristics in presumably homologous regions of the monkey, which themselves have only partially been worked out (Imig et al. 1977; Katsuki et al. 1962; Merzenich and Brugge 1973; Pfingst et al. 1977; Vaadia et al. 1982). If all or part of the left rostral parakoniocortex was spared in M.S., and if the tonotopically organized rostral field (R of Imig et al. 1977) of the monkey is functionally as well as anatomically (Galaburda and Sanides 1980) homologous to the rostral parakoniocortex in man, sharply tuned, tonotopically organized frequency information may have been available for processing; the flow of information would be limited posteriorly by the lesion but could proceed rostrally to higher order modality specific and heteromodal association areas. However, even if portions of sharply tuned, tonotopically organized regions of association cortex were spared in M.S., the principal source of such information, the primary area, was lesioned bilaterally. The afferent input to the spared auditory association areas might therefore be limited to projections from the thalamus.

The differential distribution of thalamic afferents to the various auditory fields is uncertain in man. The connectivity patterns of presumably homologous regions in the monkey (Burton and Jones 1976; Jones and Burton 1976; Mesulam and Pandya 1973; Trojanowski and [illegible] son 1975) show that the auditory association [illegible] receive direct input from the thalamus. In M.S. [illegible] may have reached the [illegible] [illegible] projections from dorsal and [illegible] the medial geniculate body (MGB) and [illegible] posterior nuclear complex. In [illegible] [illegible] capsular extension of the [illegible] [illegible] the thalamocortical [illegible] [illegible] that many of the neurons [illegible]

show inconsistent frequency responses and irregular tonotopicity in both the monkey (Gross et al. 1974) and cat (Aitkin 1973; Calford 1983; Phillips and Irvine 1979), no sharply tuned, tonotopically organized frequency information may have been available to the spared cortical association areas; at the very least, it was likely incomplete, perhaps owing to projections from sharply tuned cells in some regions of magnocellular MGB (Aitkin 1973) and lateral pulvinar (Phillips and Irvine 1979).

The corticocortical connectivity patterns of the auditory areas that appeared to be spared in M.S. may likewise be considered with regard to presumably homologous areas in the monkey (for review see Pandya and Yeterian 1985). In the right hemisphere, the intrinsic rostral connections of the spared posterior association areas with the primary area and anterior superior temporal region appear to be completely interrupted; long projections to and from prefrontal cortex may have likewise been destroyed, but reciprocal connections of the posterior association areas with the posterior cingulate gyrus and heteromodal temporoparietal regions may have been at least partially spared. In the left hemisphere, caudal connections of the spared anterior association areas were likely interrupted, but the intrinsic connections of the rostral parakoniocortex, isocortex, and proisocortex along the anterior superior temporal region and the long connections of these areas to prefrontal cortex and medial temporal structures may have been spared in their entirety. The island of preserved posterior association cortex on the left appears to be completely disconnected from the rostral primary and association areas, but its reciprocal connections with posterior cingulate gyrus and heteromodal temporoparietal cortex may have been at least partially spared.

Structure–Function Considerations

There has been some controversy in the animal literature as to whether or not bilateral lesions of auditory cortex permanently impair pure tone sensation (for review see Neff et al. 1975), but recent psychoacoustic measurements in monkeys have documented increased detection thresholds, most prominently in the mid-frequency range, as long as 1 year after extensive bilateral ablations (Heffner and Heffner [illegible]). In the neurological literature [illegible] measurements of pure tone [illegible] cortical hearing loss [illegible] in whom peripheral [illegible] be ruled out [illegible] 27 year old [illegible] thresholds [illegible] middle [illegible] -ished com- [illegible] Hirano [illegible] transverse [illegible]

reported persistent but improving hearing loss at 3 months follow-up in a 20-year-old man following successive contralateral middle cerebral artery strokes; no anatomical data were available beyond angiography and technetium brain scan. The present case was previously reported to show complete deafness 3 weeks following his second stroke and moderately increased (and variable) pure-tone thresholds 18 months out (Mendez and Geehan 1988). Apparently, recovery of sensory functions can continue and attain normal levels more than a year and a half after onset. In view of the lesion localization, the normal sensory thresholds may be attributed to sparing of the thalamus (beyond any possible retrograde degeneration not imaged by MRI), partial sparing of association cortex, or both.

Bilateral lesions of auditory cortex in animals are known to cause permanent deficits affecting the discrimination of various complex spectra, including intermittent vs. steady white noise (Symmes 1966), human vowel sounds (Dewson 1964; Dewson et al. 1969), and species-specific vocalizations (Heffner and Heffner 1988). The clinical literature contains a number of case reports in which the perception of complex tonal spectra, including musical sounds, is impaired out of proportion to hearing loss (at least overtly) following bilateral temporal lobe lesions (for reviews see Benton 1977; Hecaen and Albert 1978; Marin 1982; Vignolo 1969; Wertheim 1969). Two previous experiments are particularly relevant to the present observations. Whitfield (1980) demonstrated in the cat that the capacity to derive the harmonic "best fit" of a schedoharmonic series was permanently abolished following bilateral ablations of primary auditory cortex and part of the surrounding fields, while relative pitch discriminations based on differences between individual spectral components could be relearned. In man, Zatorre (1988) has recently reported that removal of all or part of the right transverse gyrus(i) along with all of the anterior superior temporal region commonly impairs the perception of the "missing fundamental" (Schouten 1938), which like tonal consonance perception requires the capacity to abstract constancies among the frequency relationships of simultaneous spectral components. These latter cortical regions were infarcted in M.S.

Several authors have commented on the possible correlation between the severity of auditory deficits caused by cortical lesions and lesion size within specialized anatomical zones (Allen 1945; Dewson et al. 1969; Symmes 1966). Colombo et al. (1990) have recently raised the possibility that the size of lesions limited to auditory association cortex may influence the recovery of short-term memory for pitch. In man, there is evidence that the extent of damage to the left auditory cortex predicts the recovery of single-word comprehension (Selnes et al. 1983) and sentence comprehension (Naeser et al. 1987) following middle cerebral artery stroke. In M.S., the lesions of the primary areas were complete, suggesting that spectral frequency information processing is essential to tonal consonance perception. The functional significance of the different amounts of sparing within the right and left association areas in the present case cannot be ascertained beyond the assertion that they were sufficient to sustain musical priming.

A number of confounding variables preclude broad-ranging structure-function correlations in the present case. Because M.S. suffered his second stroke 9 years ago, some reorganization of auditory functions within the spared anatomical zones may have taken place. Furthermore, the uncertainties surrounding hemispheric specialization in auditory nonverbal functions make it difficult to ascertain whether the spared posterior association areas in the right or left hemispheres, spared anterior association areas in the left hemisphere, or all the spared areas combined subserved residual functions. Our recent observation of musical priming by the right hemispheres of two split-brain patients (Tramo and Bharucha 1990) is too limited to permit generalizations concerning laterality effects in the present case. Even if laterality effects in right-handers were known, M.S.'s mixed handedness would preclude a straightforward conclusion. Finally, there are the uncertainties surrounding the interspecies comparisons entertained above, which are especially troublesome in view of the uniquely strong lateralization of auditory-verbal functions in man. The most parsimonious interpretation of the present data is that (1) the perception of tonal consonance is chronically impaired following complete bilateral lesions of primary auditory cortex combined with partial bilateral lesions of auditory association cortex; and (2) musical priming does not rely on the integrity of primary auditory cortex.

Neural Networks in Music

On the basis of the cytoarchitectonic and connectional organization of the superior temporal region in the rhesus monkey, Galaburda and Pandya (1983) have proposed that the auditory cortex comprises three parallel processing streams, the "root," "core," and "belt" lines, which are oriented rostrocaudally along the circular sulcus (Fig. 7). Within each line, cytoarchitectonic differentiation progresses caudally in a stepwise fashion across four stages. Each stage is richly and reciprocally connected to adjacent stages within each line and to its corresponding stages across lines. Pandya and colleagues have hypothesized that the existence of multiple stages of auditory representation signifies hierarchical levels of information processing, such that the rostral flow of connections from cortical layer III to layer IV mediates the successive elaboration of thalamic input and the caudal flow of connections from layers V and VI to layer I mediates the integration of limbic influences on perception (Galaburda and Pandya 1983; Pandya and Yeterian 1985). If a similar organization characterizes human auditory cortex, as Galaburda and Sanides' (1980) work

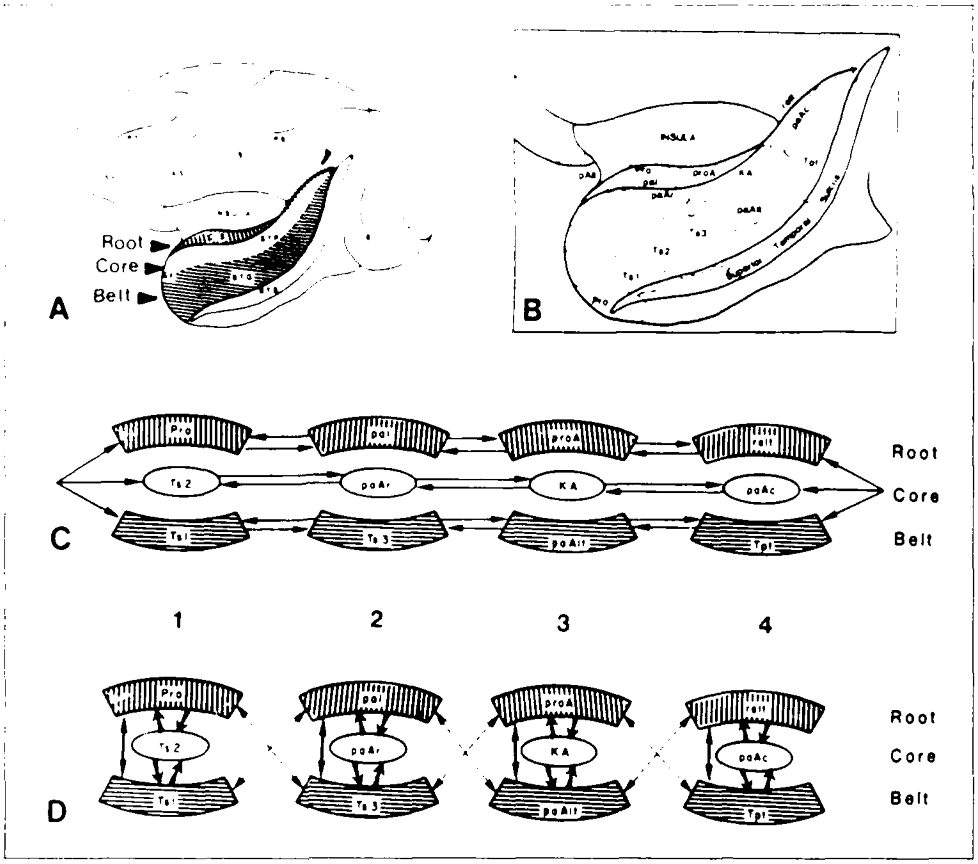


Figure 7. Anatomical model of the auditory cortex proposed by Galaburda and Pandya (1983) on the basis of cytoarchitectonic trends and connectivity patterns. See text for description. Cis, circular sulcus; KA, koniocortex; paAc, caudal parakoniocortex; paAlt, lateral parakoniocortex; paAr, rostral parakoniocortex; pal, parainsular area; pAll, periallocortex; Pro, proisocortex of the temporal pole; proA, prokoniocortex; reit, retroinsular area; STG, superior temporal gyrus; STP, supratemporal plane; STS, superior temporal sulcus; Tpt, temporoparietal isocortex; Ts1, Ts2, Ts3, rostral superior temporal isocortex. (From Pandya and Yeterian 1985. Copyright by Plenum Publishing Corp. Reprinted by permission.)

suggests, all three streams were disrupted bilaterally in M.S. by lesions within the koniocortex and adjacent association areas; stages lying at the rostral and caudal extents of each stream, which have features of isocortex and proisocortex and interact with paralimbic and heteromodal association areas, were spared to varying degrees in each hemisphere.

The hierarchical organization of auditory information processing, as it pertains to harmony perception in music, has also been modeled as a parallel distributed system on computational grounds (Bharucha 1987, Fig. 8). Three stages of processing are identified: the tone level, whose units abstract octave-equivalent pitch information; the chord level, whose units abstract more complex harmonic features; and the key level, whose units abstract even more complex harmonic features. The connectivity patterns between different stages of processing can be learned by extensive exposure to music on the basis of a number of available neural net learning algorithms, including competitive learning (Rumelhart and Zipser 1986). Once learned, the network embodies hierarchical feature relationships that can achieve (1) bottom-up ac-

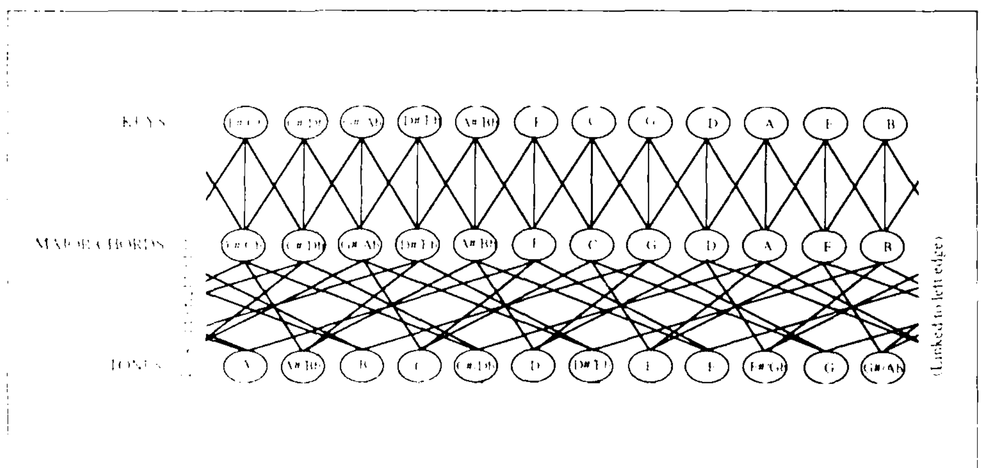


Figure 8. Computational model representing the schematic organization of tonal hierarchies in music. Letters designate scale tones within the key, chord, and tone levels of representation. Links between nodes reflect the membership of tones in chords and the membership of chords in keys. Network output creates the network from bottom-up and top-down influences over successive processing cycles (top-down). (From Bharucha 1987. Copyright by the Cognitive Science Society. Reprinted by permission.)

tivation of representations of complex features such as tone chroma, chords, and keys form the sounded tones, and (2) top-down activation of representations of chords and tone chroma from the activated keys and, consequently, the observed facilitation of chord intonation judgments. In the context of Galaburda and Pandya's anatomical model of auditory cortex, and in view of the present findings, the neural substrate for top-down influences in musical priming may be distributed within the rostral and/or caudal reaches of auditory association cortex. Given this localization, and given that cognitive representations of tonal hierarchies in music are presumably learned, it is noteworthy that frequency specific response plasticity during associative learning is a common property of neurons in the auditory association cortex of the cat (Diamond and Weinberger 1984; Weinberger and Diamond 1988); cells within both the primary and secondary fields manifest frequency specific response plasticity, and they comprise a greater proportion of the sampled population in the latter than in the former.

The existence of cortical feature detectors that are selectively responsive to musical sounds (Katsuki et al. 1962) and to the harmonic composition of sounds (Winter and Funkenstein 1973) has been previously hypothesized on the basis of neuronal responses to complex spectra in monkeys. Response selectivity to acoustic transients in human speech sounds, including fundamental frequency, has been reported (Steinschneider et al. 1982). Neurons in the secondary fields appear to be slightly more selective for complex features than those in the primary field, but a considerable degree of variability in both response selectivity and response patterns has been observed at the single cell level (Manley and Mueller-Preuss 1978; Newman and Wollberg 1973). Unit responses to simultaneous pure tones cannot be predicted with certainty even on the basis of that unit's responses to each pure tone element (Katsuki et al. 1962). This variability has led Manley and Mueller-Preuss (1978) to postulate the existence of "detection networks" whose units respond in a probabilistic rather than deterministic fashion and collectively contribute to percept formation based on the conglomerate pattern of unit discharges.

That contextual influences on chord intonation judgments remained operative in M.S. even though the cortical association areas may have been deafferented from finely tuned, tonotopically organized frequency information raises the possibility that broadly tuned subcomponents of the thalamocortical auditory system mediate musical priming. From a cognitive perspective, broad tuning is not so much a lack of fine tuning as an attribute that is well suited to a system that abstracts constancies among the spectral features of different acoustic signals and ignores small differences among elementary acoustic parameters that are insignificant to categorical processing and stimulus recognition. Given the observed interaction between incoming sensory information and prior events in musical priming, it is interesting that the responses of broadly tuned cortical auditory neurons to a given frequency are often influenced by the stimulus relationship to a prior frequency (McKenna et al. 1989; Whitfield and Evans 1965).

Of course, these parallels between anatomical, physiological, and computational models of tonal information processing must remain conjectural in anticipation of further empirical justification.

METHODS

Neurological History and Examination

The details of the neurological history have been previously published by Mendez and Geehan (1988). Briefly, M.S. is a 30-year-old man who in 1980 and 1981 suffered focal infarcts in the distribution of the right and left middle cerebral arteries, respectively, presumably secondary to cardiogenic emboli. His first stroke presented with left hemiparesis and his second with transient deafness and persistent deficits involving both speech and environmental sound perception.

At the time of the present observations, M.S. reported a pervasive sense that "everything doesn't sound clear." He had great difficulty with figure-ground separation, for example, he stated he had trouble understanding speech when more than one person was talking, and when he rode in his car with the window down he had to greatly increase the loudness of his radio in order to apprehend the music being played. His wife, a speech therapist, noted that in the face of competing sounds his speech comprehension was aided by facing him and by talking somewhat slowly.

M.S. almost exclusively listened to music sold before his strokes. He denied that music unfamiliar to him had an unpleasant, dysacusic, or dissonant quality. He had difficulty understanding the lyrics of new songs, but he remembered the lyrics (and music) of familiar old favorites well enough to recognize them when he heard them on the radio. Although he spent fewer hours per day listening to music than before his strokes, he attributed this to a change in life style rather than a lessened appreciation of music. He denied ongoing difficulty recognizing environmental sounds, and remarked that he was able to differentiate foreign car horns from American ones by their tonal quality.

The neurological examination was remarkable for dysprosodic speech, a spastic left hemiparesis, and left hemisensory loss to all tactile modalities. M.S. was alert, attentive, and highly motivated to perform well on all tasks. His speech was fluent and well articulated, and he followed spoken commands quickly and accurately. He distributed spatial attention normally. He was ambulatory with a cane and independent in most activities of daily life. Medications were limited to coumadin. The Edinburgh Inventory (Oldfield 1971) indicated mixed hand-

edness with right predominance premorbidly (Laterality Quotient = +64, Decile R.2).

M.S.'s premorbid level of musicality corresponds approximately to Grison's (1972) third level of musical culture, which lies in the middle of her classification scheme. M.S. took guitar lessons for 1 year at age 15; he was not trained in theory and had not played the guitar for several years before his first stroke. In his teens and early twenties, he was an avid listener of popular music; he accumulated a large record collection and listened attentively to music several hours a day.

Standard Audiometric and Psychometric Tests

Pure Tone Sensation Thresholds

Audiometry was performed with the subject seated in a sound-treated room (IAC). Acoustic stimuli were generated using a Grason-Stadler 16 Diagnostic Audiometer and presented through TDH-49 earphones. Detection thresholds for a 500 msec pure tone at 250–8000 Hz (Fig. 2) were established using the modified Hughson-Westlake procedure (Carhart and Jerger 1959).

Speech Perception

The Northwestern University List Number 6 (Tillman and Carhart 1966) was presented monaurally 35 dB above average pure tone threshold. A response accuracy of 90% and interear differences of 12% lie within the 95% confidence interval in normals (Thornton and Raffin 1978). M.S. repeated 4/50 words accurately in left ear trials and 23/50 in right ear trials.

Psychometric Tests

The Boston Diagnostic Aphasia Examination (BDAE; Goodglass and Kaplan 1983), Boston Naming Test (Goodglass et al. 1983), Performance Subtest of the Revised Wechsler Adult Intelligence Scale (Wechsler 1981), and the Revised Wechsler Memory Scale (Wechsler 1987) were administered in standard fashion in a quiet room. During the presentation of the auditory verbal subtests of the BDAE, the examiner took care to face away from the patient and speak at a customary rate and loudness.

Performance on the BDAE showed normal single word recognition (80/80 correct). However, M.S. made three paraphasic errors on the sentence repetition subtest, the nature of which are pertinent to the present experimental observations. He said: "The bat leaps" for "The vat leaks"; "The Chinese man had a rare emerald" for "The Chinese fan had a rare emerald"; and "They heard him speak [illegible] the radio" for "They heard him speak on the radio." The subject's substitution errors in the first two instances are far more frequent in the lexicon (Francis and Kučera 1982), suggesting that M.S. was activating acoustic representations of speech sounds via a "degraded" signal in an attempt to enhance his impoverished consonant-vowel phoneme discrimination. Consistent with this interpretation is the observation that he substituted "leaps" for "leaks" to follow his error "bat," presumably to resolve the impending semantic incongruity, yet failed to correct the open-class substitution error "from," which did not alter the meaning of the sentence.

Performance on the Boston Naming Test was within normal limits (59/60 correct), the Performance IQ was 95, and the Memory Quotient was 91. On the W.M.S. he scored below the 50th percentile on the logical memory subtests and below the 25th percentile on the visual reproduction subtests; of course, performance on the former could have been affected by impaired speech discrimination.

Musical Priming Task

Stimuli and Apparatus

The stimuli used in the present experiment have been previously described by Bharucha and Stoeckig (1987). To summarize, chord spectra were synthesized using an Apple Macintosh microcomputer. Each prime chord and "in-tune" target chord is a major triad composed of the tonic (f_0), mediant ($2^{4/12} \times f_0$), and dominant ($2^{7/12} \times f_0$) across the equal-tempered scale. "Out-of-tune" targets are major triads that are mistuned by flattening the fifth degree by a fraction of a semitone (a frequency factor of $2^{p/96}$). Chord components were sampled from a five-octave range (65.41–4186.2 Hz; A_4 = 440 Hz) with an amplitude envelope in the frequency domain, such that the lowest and highest frequency components tapered off to the threshold of hearing as determined by the Fletcher and Munsen (1933) isoloudness curves. This procedure minimizes the salience of the lowest and highest spectral components (Shepard 1964; Krumhansl et al. 1982b). The waveform of each of the 15 component tones contained the first four harmonics with equal amplitudes.

The degree of association (harmonic relatedness) between the prime chord and target chord was varied in accordance with previous experimental analyses of harmonic relatedness judgments in normal subjects (Bharucha and Krumhansl 1983; Krumhansl et al. 1982a), which corroborate theoretical accounts of harmonic structure in Western music (for review see Piston 1978). Related pairs (e.g. A^{flat} and G^{flat}) shared parent keys, but they did not share component tones. Primes were 3 seconds in duration and targets were 4 seconds in duration.

M.S. performed one block of 96 trials. Trial presentations were internally randomized by the computer. Brief pauses lasting up to 1 min were interspersed approximately every 12 trials. Each of the 12 major chords occurred four times as a prime. The prime was followed equally by each of the following target conditions: (1) in-tune related, (2) in-tune unrelated, (3) out-of-tune related, and (4) out-of-tune unrelated.

Stimuli were presented in free field in a quiet room using a Macintosh II and a Sansui A-707 amplifier and speaker system. Stimulus frequencies lay within the high end of the pure tone frequencies used to measure sensation levels, and stimulus intensity was adjusted to 60 ± 5 dB using a Quest sound level meter. Response accuracy and reaction time measurements were internally synchronized with chord target onsets.

Procedure

A training session was conducted during which feedback was given after each practice trial. The amount of mistuning was increased by a frequency factor of $2^{1/36}$ until M.S. was to get five consecutive trials correct, but it became apparent that he had difficulty with "in tune" targets, even at a factor of $2^{1/24}$, the maximal level of deviation from the equally tempered scale. We then tested him with single chords, that is, targets without primes, but he still did not reach criterion. In collecting data for statistical analysis, we decided to mistune the out-of-tune targets by a factor of $2^{1/12}$, the maximal level of mistuning.

M.S. was instructed to press one of two horizontally adjacent keys on the computer keyboard that were labeled "IN" and "OUT." He used his right hand, which was his preferred hand for fine motor control premorbidly. Power, rapid *alternating* movements, and proprioception at the fingers were normal, and there was no evidence of response disinhibition on informal "go no-go" testing.

To begin each trial, the examiner pressed the space bar on the computer keyboard. The trial began with a 2 sec mask consisting of 16 tones of random pitch, followed by a 1 sec pause, then the prime chord, then the target (Fig. 1).

Error rates were analyzed using an analysis of variance with replications as the random factor.

Spectral Intonation Judgments in the Absence of a Prime

The stimuli, apparatus, and procedure were the same as described in the priming task, except that the mask was followed by a single chord, that is, an in tune or out-of-tune major triad target without a prime. M.S. heard each target twice and performed one block of 48 trials.

Brainprints

MR scans were obtained using a Siemens 1.0 Tesla Magnetom MR System. Forty-two contiguous T1 weighted coronal sections were obtained in the coronal plane (TR = 600 msec, TE = 20 msec, slice thickness = 3.8 mm, gap = 0, acquisitions = 4, in plane resolution 1.2 × 1.2 mm). In addition, two sets of 20 T2 weighted images were obtained (TR = 2500 msec, TE = 45/90 msec, slice thickness = 6.0 mm, gap = 1.5 mm, acquisitions = 1, in plane resolution 1.2 × 1.2 mm). T2 weightings are routinely more sensitive than T1 for infarct imaging, and the TR/TE 2500/45 weighting was particularly helpful for discriminating infarcted tissue from cerebrospinal fluid within ex vacuo change. Mid-sagittal scouts were obtained at the outset to align and mark the planes of section on an internalized grid and ruler and to measure the anterior/posterior distance between the frontal and occipital poles.

The computer reconstruction method used to flat map each hemisphere was originally detailed by Jouandet et al. (1989) and will be only briefly described here, along with minor modifications adopted for pathoanatomical analysis. Copies of each T1 weighted image were placed in a Beseler 23HC photographic enlarger and projected onto paper; the pial surface of each hemisphere and the borders of the lesion cut it appeared on that sheet were traced. T2 weighted images were projected onto the closest corresponding T1 weighted tracings, from which they were 1.7–2.0 mm away, to further estimate the pial margins of necrotic tissue and the borders of abnormal signal not apparent on the T1-weighted images. Reference points demarcating individual gyri, intrasulcal vs. superficial cortex, surface structures lying within areas of abnormal MR signal, and surface structures overlying areas of abnormal signal in subjacent white matter were numbered sequentially from dorsomedial to ventromedial. The cortical convolutions on each section were labeled in accordance with the coronal atlases of Matsui and Hirano (1978) and Krieg (1963). The tracing was then entered into the computer via a Kurta IS/One digital graphics tablet for straightening, alignment, connection, scaling, and supplemental graphics. The surface areas of individual regions of interest were measured using a digitalized planimeter. Hemispheric volumes were measured by tracing the surface of each hemisphere on each slice using the planimeter, then tracing the ventricles (when necessary), subtracting the ventricular surface area from total hemisphere surface area, and multiplying by slice thickness.

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APPENDIX
Glossary of Abbreviations for Brainprint Regions of Interest

A	Amygdala	Lg	Lingual gyrus
Ang	Angular gyrus	Oi	Inferior lateral occipital gyrus
Bf	Basal forebrain	Om	Middle lateral occipital gyrus
Cn	Precentral gyrus	Or	Orbitofrontal gyri
Cg	Cingulate gyrus	Os	Superior lateral occipital gyrus
Cp	Postcentral gyrus	Pc	Precuneus
Cs	Cuneus	Ps	Superior parietal lobule
Fop	Inferior frontal gyrus, pars opercularis	R	Rectal gyrus
Forb	Inferior frontal gyrus, pars orbitalis	Sm	Supramarginal gyrus
Ft	Inferior frontal gyrus, pars triangularis	Ti	Inferior temporal gyrus
Fm	Middle frontal gyrus	Tm	Middle temporal gyrus
Fs	Superior frontal gyrus	TP	Temporal pole
Fus	Fusiform gyrus	Ts	Superior temporal gyrus
H	Hippocampal region, including the parahippocampal gyrus	U	Uncus
Hg	Transverse gyrus(i) of Heschl		

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GROSS MORPHOMETRY OF FRONTAL, PARIETAL, AND TEMPORAL CORTEX IN MONOZYGOTIC TWINS C.E. Thomas*, M.J. Tramo, W.C. Loftus*, C.H. Newton*, and M.S. Gazzaniga Program in Cognitive Neuroscience, Dartmouth-Hitchcock Medical Center, Hanover, NH 03756

We considered the role of genetic influences in the development of the cerebral cortex and regional variation therein by comparing the size and shape of the frontal, parietal, and temporal cortex of two pairs of monozygotic twins using the cortical flat-mapping technique of Jouandet et al. (J Cog Neurosci 1:88-117,1989).

All subjects were young right-handed women. Monozygosity was determined by red blood cell surface marker assays and a standardized questionnaire. Straight-line two-dimensional computer reconstructions of the unfolded cortical surface ("brainprints") were generated from thin-section T1-weighted coronal magnetic resonance images. The per cent surface area overlap of each map was determined by: 1) superimposing two maps of a given lobe aligned at the dorsomedial-most sulcus of that lobe on the brainprint (cingulate sulcus for the frontal and parietal lobes, lateral fissure for the temporal lobes); 2) tracing by hand the outer border of the superimposed maps and the region of overlap within it; 3) measuring by computerized planimetry the surface area contained within the former and the latter; and 4) calculating their per cent overlap. Maps of each lobe were paired within and across twins.

In twin pair A, left frontal, parietal, and temporal cortex showed 81%, 71%, and 79% overlap and the right 82%, 70%, and 83% overlap, respectively. In twin pair B, left frontal, parietal, and temporal cortex showed 75%, 78%, and 82% overlap and the right 79%, 71%, and 76% overlap, respectively. The mean overlap of left frontal, parietal, and temporal cortex of the unrelated twin pairings (N=4) was 82%, 71%, and 81% and of the right 75%, 67%, and 77%, respectively. The mean overlap of all pairings combined (N=12) was 79%, 70%, and 80%, for frontal, parietal, and temporal cortex.

These preliminary findings raise the possibility that the gross morphometry of parietal cortex varies more than that of frontal and temporal cortex. No striking difference was observed in twin pairs versus unrelated pairs. [Supported by ONR N00014-89-J-3035 (MJT & MSG) and AFOSR-89-0437 (MSG)]

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BRAINPRINTS: INTER- AND INTRA-OBSERVER RELIABILITY

M.L. Jouandet, M.J. Tramo, C.E. Thomas*, C.E. Newton*, W.C. Fisher, John B. Weaver*, and M.S. Gazzaniga. Program in Cognitive Neuroscience, Dartmouth-Hitchcock Medical Center, Hanover, NH 03756 and Dept. of Radiology, The New York Hospital-Cornell Medical Center, NY, NY 10021

We recently described a new in vivo method of unfolding, mapping and measuring the human cerebral cortex using straight-line two-dimensional computer reconstructions of magnetic resonance images (Jouandet et al. 1989 J Cog Neurosci 1:88-117). In order to consider the error inherent in our reconstruction procedures, inter- and intra-observer reliability were assessed by having four observers independently map one hemisphere and a fifth observer map one hemisphere three times.

Among the four observers, total hemispheric surface area (SA) ranged from 930-1067cm^2, frontal lobe SA 229-249cm^2, parietal lobe SA 219-248cm^2, temporal lobe SA 193-222cm^2, occipital lobe SA 102-145cm^2 and limbic-paralimbic cortex SA 154-160cm^2. As an index of inter-observer reliability, the coefficient of variation (CV) of each region of interest (ROI) SA measurement was calculated by dividing the population standard deviation by the population mean. In addition, pair-wise correlations across ROI SAs for the four observers were calculated. For total hemisphere, frontal, parietal, temporal, and occipital SA, CV was 5.4%, 3.5%, 6.4%, 2.7%, and 15%, respectively. Among the 27 ROIs, SA measurements showed the least variation in the orbitofrontal gyri and pars triangularis (3.0%) and the greatest in the superior lateral occipital gyrus (28%). ROI median CV was 9.4%. Pair-wise correlations ranged from .95-.99. CVs were also calculated to estimate intra-observer reliability. For total hemisphere, frontal, parietal, temporal, and occipital SA, CV was 2.7%, 2.1%, 3.7%, 4.4%, and 8.3%, respectively.

These results compare favorably with accuracy estimates of regional cortical SA measurements obtained using the contour method of flat-mapping post-mortem animal cortex (Van Essen and Maunsell 1980) and indicate that our brainprinting technique yields reasonably reliable estimates of regional cortical SA in vivo.

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MARC L. JOUANDET (212)-746-2548

APPENDIX III

New Statistical Methods for Evaluating

Effects of Redundant Signals on Response Times

Georgie Nozawa

Department of Psychology

Dartmouth College

This paper presents statistical methods which can be used to examine two types of inequalities obtained in paradigms using redundant targets. These methods have been developed to analyze the latency data obtained in our investigation of multimodal control of orienting. In the redundant target paradigm (see for example, Egeth, Jonides, & Wall, 1972), a certain number of identical targets are presented simultaneously to the subject. The subject's task is to detect, recognize, or identify the target. In general as the number of redundant targets increases, error rates and detection latencies decrease monotonically (Grice, Canham, & Boroughs, 1984; Miller, 1982, 1986;Nickerson, 1973; Raab, 1962; Ulrich & Giray, 1986; Shaw, 1982; Mulligan & Shaw, 1980; Townsend & Ashby, 1983).

The models which successfully account for the performance in the redundant target paradigm can be classified as parallel processing models and include probability summation or horse race models and neural summation models. The probability summation model processes the input information from different channels at the same time. The processing is terminated whenever a target has been encountered. If there are two targets to be processed, the processor will end the execution when the first target is processed. Therefore, reaction times of two targets condition is expected to be faster than reaction times to each individual target. The neural summation model which we examine here entertain another possibility. It assumes that each processing channel has an independent neural counting process. When two channels are activated at the same time, the activities of the two counting processes are summed. This summation process produces an increase in neural counting activity which leads to a decrease in times to reach a response criterion. Nozawa (1988) proved mathematically that the neural summation model is in fact faster than the horse race model or the unlimited capacity independent parallel model .

Two inequalities can be used to distinguish between horse race and neural summation models (Ulrich & Giray, 1986;Nozawa, 1988). One of these was introduced by Miller(1982) and is refered to as Miller's inequality. The second inequality is introduced in the present paper, and shall be refered to as the Survivor measure or S-measure.

Miller's inequality (Miller, 1982) can be expressed as follows:

$$P(RT \le t \mid S_1 \text{ and } S_2) \le P(RT \le t \mid S_1) + P(RT \le t \mid S_2). \qquad (1)$$

The term, $P(RT \le t \mid S_1 \text{ and } S_2)$ represents the cumulative distribution function of the redundant target condition. The other terms represent the cumulative distribution functions of single target conditions. The inequality above comes from the fact that the cumulative distribution of the redundant target condition can be expressed as the sum of two cumulative distribution functions from single target conditions minus the joint cumulative distribution function of single target conditions.

$$P(RT \le t \mid S_1 \text{ and } S_2) = P(RT \le t \mid S_1) + P(RT \le t \mid S_2) - P[(RT \le t \mid S_1) \text{ and } (RT \le t \mid S_2)]. \qquad (2)$$

The relationship between the lefthand side and the righthand side of the identity (2) leads us to the inequality used in the S-measure, which comes from the equivalent identity in terms of survivor functions. The S-measure is defined as follows:

$$\text{S-measure} = P(RT>t \mid S_1 \text{ and } S_2) - P(RT>t \mid S_1) \times P(RT>t \mid S_2). \qquad (3)$$

Ulrich and Giray (1986) showed that performance of the independent probability summation model must fit into the interval between $P(RT\leq t \mid S_1) + P(RT\leq t \mid S_2) - P[(RT\leq t \mid S_1) \text{ and } (RT\leq t \mid S_2)]$ and $\max(P(RT\leq t \mid S_1), P(RT\leq t \mid S_2))$. In terms of survivor functions the above interval can be translated into the interval between $P(RT>t \mid S_1) \times P(RT>t \mid S_2)$ and $\min(P(RT>t \mid S_1), P(RT>t \mid S_2))$.

As shown by Ulrich and Giray (1986) the cumulative function of any redundant target condition must fall between the region surrounded by two boundaries: the upper bound is the sum of the two cumulative distribution functions from single target conditions and the lower bound is the maximum of the two single condition's cumulative distributions (see Fig 1). Notice that in terms of the distribution functions, the faster the mean reaction time the greater the cumulative distribution function. So the upper bound shows us the case in which the speed of the probability summation model is maximized. On the other hand the lower bound shows us the case in which the speed of the probability summation model is the slowest possible. Increase or decrease of reaction times inside the boundaries can be explained by stochastic dependence between the processing channels as was put forth by Ulrich and Giray (1986) and Colonius (1986). However, any point beyond this region cannot be explained by any type of probability summation model that assumes stochastic dependence.

In order to reject the probability summation model and support a neural summation model, it is necessary to violate identity (2) and inequality (1): the identity (2) being a weaker test for the neural summation model and the inequality (1) being the strongest test. In terms of statistical hypotheses, inequality (1) and identity (2) can be expressed as follows.
Identity (2) is exressed in terms of survivor function and written

$$H_0: P(RT>t \mid S_1 \text{ and } S_2) - P(RT>t \mid S_1) \times P(RT>t \mid S_2) \geq 0$$

and

$$H_1: P(RT>t \mid S_1 \text{ and } S_2) - P(RT>t \mid S_1) \times P(RT>t \mid S_2) < 0.$$

Inequality (1) is translated into statistical hypotheses as

$$H_0: P(RT\leq t \mid S_1 \text{ and } S_2) - P(RT\leq t \mid S_1) + P(RT\leq t \mid S_2) \leq 0$$

and

H_1:P(RT≤t |S_1 and S_2) - P(RT≤t |S_1) + P(RT≤t |S_2) > 0.

It is possible to calculate the alpha level for the above statistical hypotheses at any point of time. It is also possible to summarize multiple comparisons by a multiple comparison alpha. By calculating the alpha level we can distinguish the various degrees of neural summation for any experimental conditions.

Application to Multimodal Experiments

In the present investigation, we are interested in comparing the degrees of neural summation in various response modes: saccadic responses, directed manual response and simple manual reaction times. These following statistical hypotheses are to be tested.

H_0:$\mathbb{S}_{Sac}(t) - \mathbb{S}_{Joy}(t) \geq 0$

and

H_1:$\mathbb{S}_{Sac}(t) - \mathbb{S}_{Joy}(t) < 0$

H_0:$\mathbb{S}_{Sac}(t) - \mathbb{S}_{But}(t) \geq 0$

and

H_1:$\mathbb{S}_{Sac}(t) - \mathbb{S}_{But}(t) < 0$

H_0:$\mathbb{S}_{Joy}(t) - \mathbb{S}_{But}(t) \geq 0$

and

H_1:$\mathbb{S}_{Joy}(t) - \mathbb{S}_{But}(t) < 0$

$H_0: M_{Sac}(t) - M_{Joy}(t) \geq 0$

and

$H_1: M_{Sac}(t) - M_{Joy}(t) < 0$

$H_0: M_{Sac}(t) - M_{But}(t) \geq 0$

and

$H_1: M_{Sac}(t) - M_{But}(t) < 0$

$H_0: M_{Joy}(t) - M_{But}(t) \geq 0$

and

$H_1: M_{Joy}(t) - M_{But}(t) < 0,$

where $S_\cdot(t)$ represents S-measure at time=t and $M_\cdot(t)$ represents the values derived from Miller's inequality at time=t. The subscripts represent various types of response modes: "Sac" represents saccade, "Joy" represents joystick, and "But" represents button press.

Now let us consider the statistical nature of the identity (2) and the inequality (1). Nozawa (1988) considered a point on a survivor functions as a realization of a binomial random variable and used this idea to test the superadditivity of the redundant target condition at the level of the survivor functions. The same approach can be applied to the above problem: that is, binomial random variables can be used to represent the cumulative distribution functions. Any number of events in a bin of a cumulative distribution function can be considered as a particular realization of a binomial random variable: $Bin(n,p) = {}_nC_k \times p^k \times (1-p)^{n-k}$, where n is the total number of points

in the sample, k is the number of events falling into the particular bin, and p is the value in the cumulative distribution function. Note that we can also represent a survivor function by a parameter p. In order to create the statistical distribution for Miller's inequality it is necessary to sum the two binomial random variables. The sum of the two single target condition's cumulative distribution functions are considered as a convolution of two binomial random variables:

$$k_1 \sim f_{k_1}(k_1) = \binom{n}{k_1} \times p_1^{k_1} \times (1-p_1)^{n-k_1},\ k_2 \sim f_{k_2}(k_2) = \binom{n}{k_2} \times p_2^{k_2} \times (1-p_2)^{n-k_2},$$

$$\text{and } k_1+k_2 \sim f_{k_1}(k_1) * f_{k_2}(k_2) = \sum_{k_2=0}^{k_1} f_{k_1}(k_1-k_2) \times f_{k_2}(k_2)$$

$$= \sum_{k_2=0}^{k_1} \binom{n_1}{k_1-k_2} \times \binom{n_2}{k_2} p_1^{k_1-k_2} \times p_2^{k_2} \times (1-p_1)^{n_1-k_1+k_2} \times (1-p_2)^{n_2-k_2}.$$

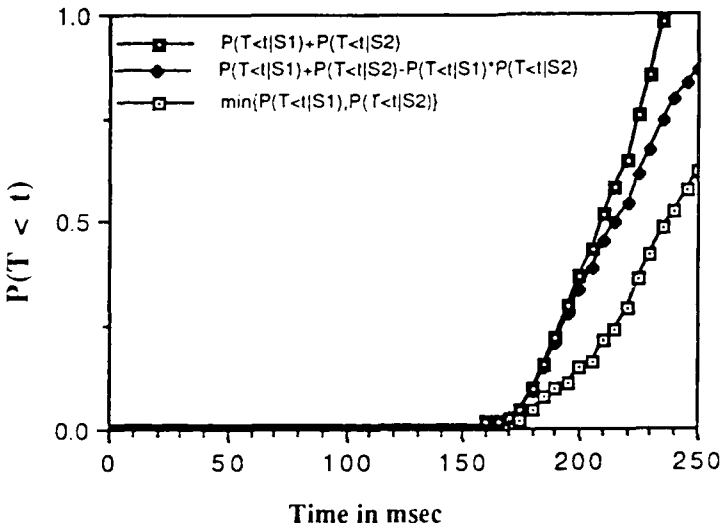
In the case of S-measure the multiplication of two single target condition's survivor functions are considered as the multiplication of two binomial random variables.

$$f_{k_3}(k3) = \sum_{k_1=0}^{\infty} \sum_{k_2=0}^{\frac{k_3}{k_1}} f_{k_1}(k_1) \times f_{k_2}(k_2) = \sum_{k_1=0}^{\infty} \sum_{k_2=0}^{\frac{k_3}{k_1}} \binom{n_1}{k_1} \times \binom{n_2}{k_2} p_1^{k_1} \times p_2^{k_2} \times (1-p_1)^{n_1-k_1} \times (1-p_2)^{n_2-k_2},$$

where $k_3 = k_1 \times k_2$.

In summary new methods to test inequalities used to distinguish probability summation and neural summation models in the redundant target paradigm were developed by treating survivor functions and cumulative distribution functions as binomial random variables.

Illustration of the regions pertaining to horse race versus neural summation models



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